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OTOLARYNGOLOGY

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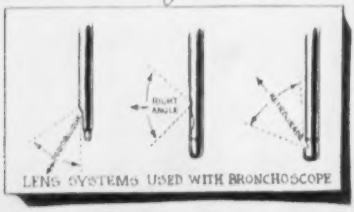

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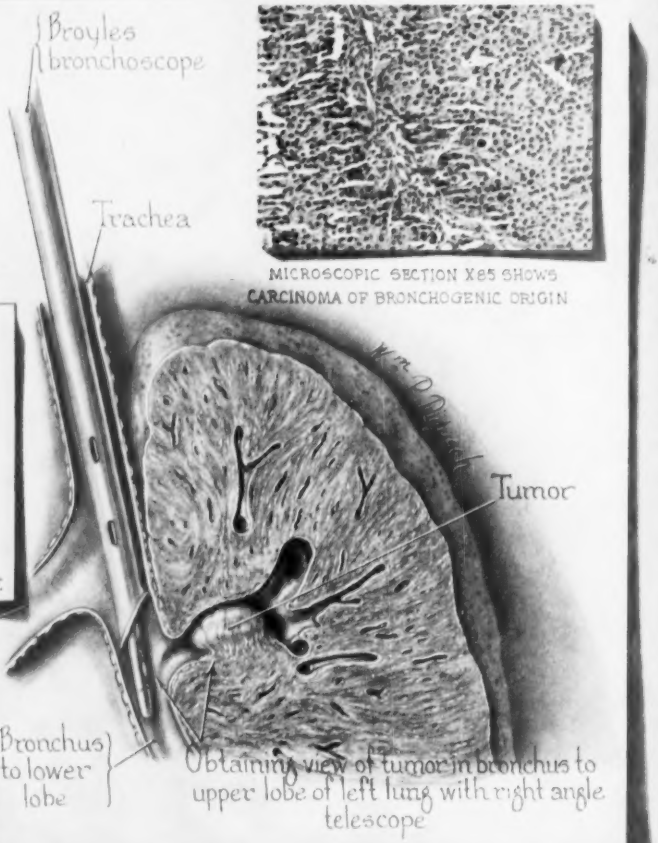
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ARCHIVES OF OTOLARYNGOLOGY

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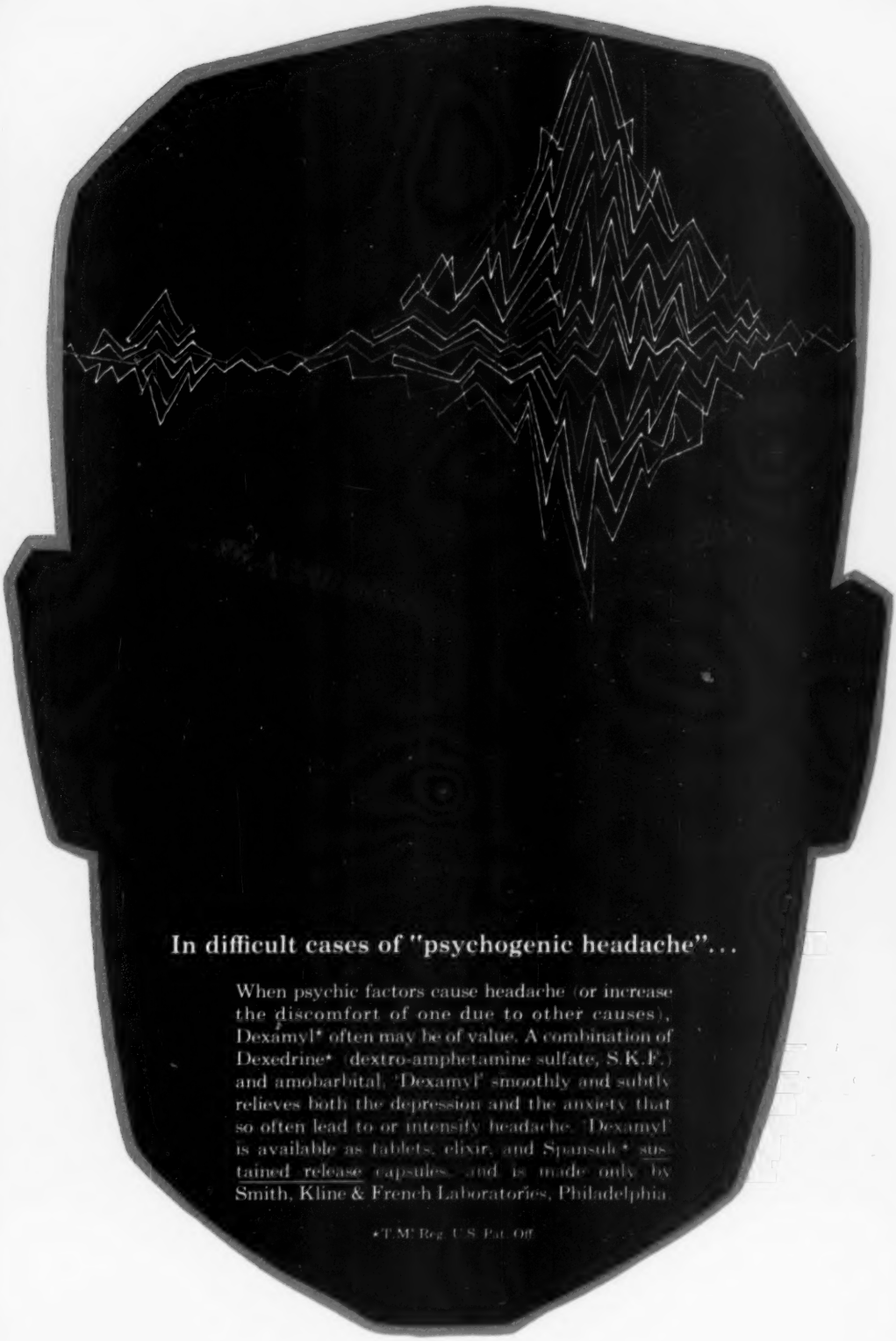

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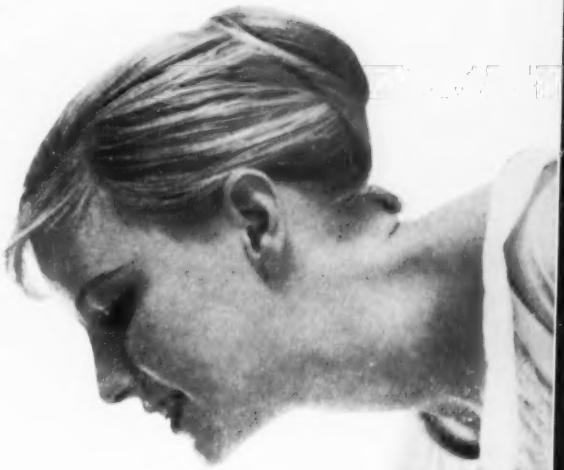

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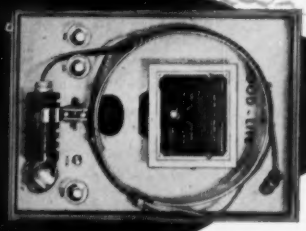

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Changing Concepts in Bronchology

ARTHUR J. CRACOVANER, M.D., New York

Those of us who have been working for a number of years in the field of peroral endoscopy have witnessed many changes in the development of this specialty. Some of these changes have been in the form of improvement in technique and instruments and a focusing of indications for the procedure. The advent of antimicrobial therapy and the advancements of thoracic surgery have produced newer and different concepts in the development of bronchology.

I have just used the word bronchology. This in itself denotes a change. Instead of acting as technicians in performing endoscopy, we now practice a specialty that goes beyond the mere performance of a procedure and the description of what is seen. A dictionary states that bronchology is "that special branch of medicine dealing with the study and treatment of the bronchial tree by means of bronchoscopy." But in order to do this we must be acquainted with the diseases and the anomalies of the entire contents of the chest. We cannot interpret the findings in the bronchus or esophagus unless we are well informed in the spheres relegated to the chest physician and surgeon as well as the gastroenterologist and radiologist. The bronchologist's scope must reach beyond the bronchial tree and the esophagus.

As the influence of the bronchoesophagologist has extended to other specialties, so have those in these specialties shown interest in bronchology and made inroads into this field. Whereas at first the endoscopist was an otolaryngologist, today the chest specialist and thoracic surgeon are participating actively in this specialty and rightly so, since it is proper and necessary that they know what is taking place within the bronchus or esophagus as well as interpreting the history, physical signs, and x-rays. But this has created difficulties in training. The otolaryngologist is supposed to remove foreign bodies from the bronchus and esophagus, and yet he is unable to obtain sufficient experience to make him proficient in this undertaking. Here is a problem that must be solved. The only answer at the moment is to relegate the responsibility to each community according to the availability of the training that is necessary.

The armamentarium has improved. Not only are instruments available to suit the individual taste, so to speak, but the visual aids in the form of improved lighting and magnification as well as the possibility of "looking around the corner" with the telescopic lenses have enhanced our ability to make a diagnosis.

The development of photography in this field—which in no small measure has been due to the efforts, interest, and foresight of Dr. Paul Hollinger—has helped greatly in teaching our subject. These photographic techniques have been adopted in other fields so as to record physiology and pathol-

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Presented before the International Broncho-Esophagological Association at its meeting in Vienna, Aug. 31, 1956.

ogy. The photographs and motion pictures aid remarkably not only in teaching members of our specialty but also in demonstrating to those on the periphery the importance of what we are able to accomplish.

The development of thoracic surgery, with the help of improved techniques in anesthesia and the discovery of antibiotics, has altered greatly the concepts in peroral endoscopy. We no longer encounter the number of cases of lung abscess we did some years ago. Pneumonia and pneumonitis are treated immediately with antibiotics before they develop into abscesses. If an abscess does exist, it may yield to the administration of antibacterial agents. In the case of a putrid abscess or a more chronic abscess the thoracic surgeons take over. The bronchoscopist is called upon to rule out bronchial obstruction due to foreign body, endobronchial disease, tumor, or an extrabronchial growth. Treatment of lung abscess by bronchoscopy is no longer in vogue, yet aspiration of the bronchial tree and instillation of the appropriate antibiotic together with systemic use of antibiotics may still give good results and should not be abandoned.

I do not come across the number of cases of bronchiectasis that I formerly did. This, too, is due to antibiotic therapy and to early bronchoscopy to eliminate bronchial obstruction. Also, the cases are recognized sooner and are localized enough to be treated surgically. Bronchoscopic clinics were crowded with patients that required frequent aspirations and lavage to reduce the amount of suppuration. It is only the patient with generalized bronchiectasis that requires such attention, and then the number of treatments are reduced by the use of proper antibiotics both systemically and through the bronchoscope.

Certainly the concept of the treatment of tuberculosis has changed. At first there was hesitation in doing bronchoscopies on cases of tuberculosis. Later it was found that bronchoscopy did no harm, and in some institutions all cases of tuberculosis were bronchoscoped and endobronchial lesions, when discovered, were treated locally. However, the treatment has been greatly altered by the discovery of chemotherapeutic and antibiotic agents. In former days there were a great many tuberculous endobronchial ulcers, granulomas, and strictures that were treated bronchoscopically. Now these frequently yield to treatment with drugs or to surgery. Bronchoscopy is still important in the diagnosis of an endobronchial lesion and to determine the condition of the bronchi before any form of therapy —surgical or collapse—is undertaken. It also is useful to ascertain the state of the bronchus during therapy. However, there are strains of tubercle bacilli that are resistant to antimicrobial agents. Also, a great many endobronchial lesions that we see today are due to compression or perforation of the bronchus by tuberculous lymph nodes. These cases may be treated locally by bronchoscopy. Any obstruction of the bronchus in tuberculosis must be investigated, and not infrequently a carcinoma is revealed.

It is in the diagnosis of carcinoma of the lung that bronchoscopy is becoming increasingly important—important because the thoracic surgeon has shown us that if we can diagnose the case early enough he can obtain a cure. We, therefore, as bronchologists must be meticulous in our examination of the bronchial tree and be suspicious of any area that shows the slightest abnormality. We must not hesitate to take many specimens—biopsies, curettings of the mucosa, washings, and smears—anything that will help to prove the presence of a carcinoma when suspected by x-ray or by symptoms. The great increase in the number of cases of cancer of the lung makes our specialty an all-important adjunct to the treatment of this dreaded disease.

The treatment of adenomas of the trachea and bronchi has been influenced by added knowledge of their pathology. Since they are known to spread beyond the limitations of the bronchial tree, only an occasional

case is treated by the bronchologist. The others undergo surgery for removal of the area involved.

There are other instances where advances in diagnosis or treatment have been augmented by the bronchologist: the diagnosis of congenital anomalies in children, the treatment of bulbar poliomyelitis, and the treatment of asthma, to name a few. Bronchography has improved, and the use of standardized terminology together with the recognition of the bronchopulmonary segments by x-ray and by bronchoscopy has been of the utmost help to the thoracic surgeon.

I do not wish to give the impression that the usefulness of bronchoscopy has diminished in any way. The indications as far as diagnosis is concerned have not changed. Its importance in treatment has been altered, but it is none the less useful and significant.

103 E. 78th St.

Cold and Collar Effects on Nerve Tissue to Simulate Bell's Palsy

J. W. BURKEY, M.D.; R. E. BRIDWELL, M.D., and G. O. PROUD, M.D., Kansas City, Kan.

Introduction

Bell's palsy poses a challenge both to the clinician and the investigator. This syndrome is generally stated to be due to a peripheral "lesion" of the seventh cranial nerve, causing typical paralysis. While the clinical signs of such nerve paralyses usually regress, in some cases they do not. Decompression of the nerve has at times brought about a return of function.[1] Better understanding of the mechanism of Bell's palsy is needed in order to better evaluate medical and surgical procedures which might be of the most benefit.

Clinical observation has long recorded a relationship of this syndrome to exposure to cold in about 70% of cases. The unique position of the facial nerve in passing peripherally through an unyielding bony canal in an "exposed area" is also pertinent. This study of the damage to nerve tissue caused by cold and the additional effect of a snug collar on nerves damaged by cold was made to estimated these factors in relation to the production of Bell's palsy.

Review of Literature

Lichtenstein[2] lists excessive cold and compression as two of several traumatic causes of neural degeneration. Kettel[3] states that facial nerves involved in clinical Bell's palsy, as seen at the time of decompression, usually show swelling. Hilger,[4] in a clinical study of Bell's palsy, believes that the neural failure is due to ischemia caused by vascular change. He considers edema to be secondary to the vascular change and not the primary cause of the ischemia.

Accepted for publication Oct. 12, 1956.

From the Department of Otorhinolaryngology of the University of Kansas School of Medicine.

Research on the pathology of trench foot[5] has shown, in sections of nerves from limbs long exposed to cold, degeneration of axis cylinders and of myelin. This degeneration is noted to be spotty. Perineural fibrosis is also noted.

Denny-Brown and Brenner[6,7] have paralyzed nerves with compression, by tourniquet and by direct pressure. They note that compression of a nerve, to cause loss of nerve function, has to be equal to the systolic blood pressure. More pressure than this does not cause more or faster paralysis. Motor nerves appear to offer less resistance to increased pressure and ischemic changes than do sensory nerves. After pressure to the nerves experimentally had caused clinical change, myelin-sheath damage lasted six to eight weeks, although function returned sooner.

The article by Kettel[3] includes a report of a rare instance in which the affected nerve of a patient with a history of facial nerve paralysis for 14 years was obtained, at death, for histological section. The pathologist's report states, in part: "The lumen of a small venous branch contains bulging, partially connected, slightly chalky connective tissue masses suggesting thrombin in full organization. Nerve filaments stain slightly, and are split by edema. Capillaries are highly dilated with blood. Small fresh punctiform hemorrhages are seen here and there, along with small groups of lymphocytes and small cicatricial strands. Matton stain shows degeneration of medullary sheath with staining of axis cylinders which appear distinctly disintegrated. Between the nerve and the sheath there is edema with fibrin."

Experimental Method

The sciatic nerve of the dog was chosen as the test material. Under intravenous thiopental anesthesia, both the nerves were exposed, and carefully freed from surrounding tissue for a distance of about 2.5 cm.

The cold-temperature factor was introduced by holding ice against the nerves for 10 minutes. Simulation of the facial nerve canal was accomplished by encompassing the nerve in a section of polyethylene tubing for a distance of 2 cm. The tubing was secured firmly but not tightly around the neural trunk with silk ties.

The usual procedure involved placing the collar around one of the two nerves, and applying ice to both nerves. After this the thigh muscles were allowed to close over the nerve, and the skin incision was sutured. The animals were caged and observed from time to time for a sign of foot lag.

The nerves were taken from the animals under general anesthesia from one to six weeks later. Both "control" and "experimental" nerves were sent to the pathologist, with separate specimens for cross and for longitudinal section. Nerves were removed from the dogs with a minimum of trauma, cutting with a Bard-Parker blade against cork.

A preliminary series of nine animals was studied with the specimens preserved in 10% formalin for hematoxylin-eosin and Bodian nerve-fiber stains. Evaluation of this material showed that a special stain for myelin was needed; therefore, a second series, of 12 dogs, was studied. The nerve specimens from these animals were preserved in 1% calcium chloride in 10% formalin. Sudan black B myelin-sheath stain was used. Spielmeyer (myelin) stain was also employed in one case.

In this second series one normal sciatic nerve was obtained as a control of all other variables. Nine animals were studied by the method described above. As a variant (two cases), both nerves were collared and only one of the nerves iced.

The pathologist evaluated the material without checking as to which procedures had been carried out on the nerve being examined in order to minimize the role of prejudice.

Both series of nerves were studied, but the greater emphasis was placed on the second series. An arbitrary scale for grading the nerves was used:

- 0 No change.
- 1+ Minor loss of staining intensity. Widening of axis cylinder space, with or without macrophages.
- 2+ Scant scattered, totally or nearly totally demyelinated fibers.
- 3+ Many totally demyelinated fibers.
- 4+ Total demyelinization.

Each slide was evaluated at various times; the grading represents a cumulative impression of the material.

Results

The results of the second series of animals as noted by the pathologist are shown graphically. It can be quickly seen that there was little difference in the findings between the collared and the "control" nerve. In both nerve series the longitudinal sections show neural degeneration to a greater degree than the cross section slides. Length of time of collaring does not appear to change the results.

A large amount of perineural inflammation and scar tissue was always present on reexposing the nerves, especially around the collared nerves. Nerve tissue on removal

Pathologist's Evaluation of Material from Microscopic Study of Second Series

Experimental Data		Pathologist's Findings				Explanatory Data
		"Control Nerve"		"Experimental" Nerve		
Animal Number	Test Time in Weeks	Cross Section	Long Section	Cross Section	Long Section	
61	1	1+	3+	1+	2+	
65	3	0	2+	2+	2+	
137	4	0	1+	0-3+	1-3+	Both nerves iced, with collar only on experimental nerve.
63	4	1+	3+	1+	2+	
67	4	1+	3+	1+	2+	
50b	4	2+	1-2+	1+	3+	
106	5	1+	2+	0	1+	*Spielmeyer stain findings.
5	6	0-2+	2+	2+	3+	
144	6	1+	2+	1+	3+	
63*	4	—	2+	1+	0-1+	
140	5	0-1+	2+	0-1+	1+	Both nerves collared; ice only to experimental nerve.
141	6	1+	1+	1+	1-2+	
50	—	0	1+	—	—	No procedure; "normal" nerve.

*Sudan black B stain was used except as noted.

from the animals usually appeared grossly swollen.

On clinical observation of the dogs during both test series, foot lag was noted to be present only sporadically, and was present in legs without collared nerves as often as in legs with collared nerves.

Comment

There is lack of correlation of clinical evidence of damage to the nerves with the anticipated greater damage to nerves which have been collared as well as iced. This is based on the assumption that the sciatic nerve controls the leg muscles, and the observation that after the nerve is sectioned the dog no longer has use of the leg.

A lymph node alongside a nerve which had been collared and iced (Dog 137) showed deposit of fat, proving that demyelinization had taken place.

The scar tissue which was present around the nerves could account for some of the neural damage, as could the mere handling of the nerve. The "snugness" of the collar on the nerves varied somewhat with the size of the nerve. This factor is not thought to be of great importance, since both nerves on any one dog were of approximately the same size.

Pathologist's Comment

Evaluation of material obtained from the first set of dogs permitted few conclusions concerning nerve injury. One observation, however, was relatively constant. A subacute inflammatory reaction was induced in the region of the plastic collar rather promptly and with great regularity. Although a scattering of polyps could be found within these lesions, no evidence of suppuration was detected; and the reaction promptly gave way to one in which lymphocytes, large mononuclear leucocytes, and fibroblasts predominated.

Examination for neural injury in the same material with the use of nerve-fiber stain proved to be disappointing, showing little evidence of fiber disease. In only one instance was there evidence of nerve-fiber damage, this occurring at the site of collaring and consisting of fragmentation with nodulation and clubbing. In every other instance the nerve fibers showed little evidence of alteration with the questionable exception of swelling, which occurred in both collared and uncollared funiculi.

More consistent alterations were found in the sections from the second series, in material stained for myelin using Sudan black B. This appears to be a satisfactory stain for use in evaluating this material.

It is felt that nerve damage estimated as less than 3+ is probably of little significance and possibly subject to error in interpretation. It will be noted that the control animal, Dog 50, showed 1+ demyelinization in a longitudinal section (Figs. 1 and 2).

It is apparent that cross sections of nerves are totally unsuitable for evaluation, since the demyelinization is patchy and irregular, and could only be appreciated adequately if longitudinal sections were available. This variation in nerve injury has been observed previously by Friedman[5] in his studies on trench foot.

Examination of the data derived from this study would seem to indicate that minimal nerve damage was induced by the procedure

Fig. 1.—Longitudinal section; reduced 1/5 from mag. × 500. Sudan black B stain. Control nerve showing 1+ demyelinization.

Fig. 2.—Longitudinal section; reduced 1/5 from mag. × 500. Sudan black B stain. Experimental nerve showing 3+ demyelinization.

of nerve collaring alone. This did not exceed the estimation of 2. In only three of the nine dogs studied did the addition of cold injury to the collared funiculus result in damage of greater degree. The application of cold alone yielded results comparable to simple collaring and collaring with the addition of cold.

Conclusions

1. The very slightly over-all greater damage shown by the nerves which were both iced and collared as compared with those controls which were only iced is not believed to be statistically significant.

2. Such findings do suggest, however, that collaring of a nerve does not enhance injury due to cold.

3. The general absence of nerve paralysis in the animal does point out the difficulty of simulating Bell's palsy even with ice placed directly on a snugly collared nerve.

Summary

This paper deals with the two factors thought to produce peripheral facial paralysis. Under controlled conditions these factors of cold temperature and rigid collar effect were explored experimentally. The changes, thereby, produced are discussed.

Dr. F. A. Mantz, of the Department of Pathology and Oncology, aided in the interpretation of the microscopic material.

University of Kansas Medical Center. (12)

REFERENCES

1. Cawthorne, T.: Discussion on Limitation of Operative Treatment for Facial Paralysis, Proc. Roy. Soc. Med. 34:582, 1941.

2. Lichtenstein, B. W.: Textbook of Neuropathology, Philadelphia, W. B. Saunders Company, 1949, p. 50.

3. Kettel, K.: Bell's Palsy: Pathology and Surgery, Arch. Otolaryng. 46:427, 1947.

4. Hilger, J.: The Nature of Bell's Palsy, Laryngoscope 59:228, 1949.

5. Friedman, N. B.: The Pathology of Trench Foot, Am. J. Path. 21:387, 1945.

6. Denny-Brown, D. E., and Brenner, C.: Paralysis of Nerve Induced by Direct Pressure and by Tourniquet, Arch. Neurol. & Psychiat. 51:1, 1944.

7. Denny-Brown, D. E., and Brenner, C.: Lesion in Peripheral Nerve Resulting from Compression by Spring Clip, Arch. Neurol. & Psychiat. 52:1, 1944.

Tracheal Diverticulosis

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Tracheal diverticula are rarely diagnosed clinically and are usually only discovered at autopsy. Rokitansky [1] was one of the first to describe these lesions accurately, and it was his belief that they developed as a result of chronic pulmonary and bronchial infection. He stated that they occurred in the upper part of the right posterior tracheal wall, and that they originated from mucous glands. Chiari,[2] however, contended that some diverticula were rudimentary bronchi, occurring on the right side of the trachea at the site of tripartition of the bronchial tree. In 1889 he described six autopsies in which tracheal diverticula were present on the lower end of the trachea on the right between the 17th and 22nd tracheal rings. This type of growth always extends obliquely down and outward, the shape being that of a short, cylindrical tube.

Ziegler and Kahlden, in 1897,[3] and Mackenzie, in 1911,[4] were among the first to publish case reports of these lesions.

McKinnon [5] described 10 cases diagnosed at autosy, 8 of which were found in 867 routine autopsies. The diverticula were attached to the right posterior-lateral border of the trachea in every instance. In seven of the cases only one diverticulum was present, but in other cases two diverticula were discovered. Histological study revealed the sacs to be lined by stratified ciliated columnar epithelium, and to have arisen from mucous glands.

Drymalski et al.[6] report a case diagnosed at autopsy in a 66-year-old man with bronchiectasis. Two tracheal diverticula were present on the right posterior-lateral aspect of the trachea approximately 2.5 cm. distal to the larynx. Each communicated with the trachea through a tiny orifice.

Stibbe [7] reported a case in a 46-year-old woman in which the diverticula had a pinpoint orifice about 1 cm. above the bifurcation. The patient gave no history of respiratory disease, but she had a right aortic arch malformation.

Kahlstrom [8] demonstrated a series of these lesions with iodized oil (Lipiodol) in a patient with syphilitic aortitis, aneurysm of the arch, and bronchiectasis.

Nielsen [9] reported a diverticulum-like protrusion of the posterior wall of the upper part of the trachea, associated with tracheal widening and dilatation of the bronchial tree, in a patient with a long history of respiratory disease. He states that acquired diverticula have a narrow inlet, are often multiple, and usually contain viscid mucus.

Kernan [10] attributes the death of a 2-month-old infant to a diverticulum which eroded into the innominate artery.

Addington, Rusk, and Cohen [11] reported a case in a 32-year-old man with bronchiectasis. The upper one-third of the trachea was dilated, and an air-filled 4×5 cm. diverticulum was present in the right posterior-lateral wall of the trachea. The esophagus was displaced to the left and moderately narrowed.

Miller [12] described the trachealis muscle as being composed of transverse bands, and stated that between these bands there was a relatively amuscular area which served as the site of election for the development of a diverticulum. Other probable factors important in the development of acquired diverticula are infection in the mucous glands of the posterior tracheal wall and increased intratracheal pressure (as from a persistent cough).

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Figure 1A

Figure 1B

Figure 2A

Figure 2B

Report of a Case

The patient, a 43-year-old white man, was admitted to San Francisco City and County Hospital on Sept. 9, 1956, complaining of having a "chicken bone caught in the throat." The past history and review of systems were unremarkable except for the presence of a nonproductive cough for the previous four years. This cough was most annoying in the early mornings for a period of about 20 minutes. Physical examination revealed the patient to be in excellent condition except for the fact that he was edentulous. X-rays revealed a radiopaque object measuring 2 mm. by 3 cm. parallel to and between the trachea and the cervical spine at the level of C 6 (Figs. 1A and 1B). Immediately below the foreign body on the lateral film a radiolucency measuring 2×1 cm. was noted. The trachea was widened in its midportion. A barium swallow was done, but no esophageal diverticula were found. However, the esophagus was noted to deviate in a smooth curve to the left of the midline at the level of T 1 and T 2, apparently because of the tracheal widening (Figs. 2A and 2B). The foreign body was removed at esophagoscopy and proved to be a piece of bone. Subsequently, tracheograms were done (Figs. 3A and 3B).

The above-mentioned 2×1 cm. radiolucency represented a diverticulum of the trachea, with a relatively narrow inlet; in addition, the contrast material (an iodopyracet derivative [Oily Dionosil]) outlined numerous other diverticula, located on the right posterior aspect of the trachea and varying in size from 3 mm. in diameter to the 2×1 cm. air-filled diverticulum seen on the plain films.

Figure 3A

Figure 3B

The patient was discharged from the hospital in good condition. He returned several days later with a faint urticarial reaction, presumably from penicillin received while in the hospital. This reaction cleared rapidly on antihistamine therapy. At the time of readmission another film was taken to determine if any opaque medium remained in the diverticula; none was demonstrated.

Comment

Because of the configuration of the diverticula, with a relatively narrow inlet, and their location on the right posterior-lateral wall, it would appear that these lesions are of the acquired type. On the preliminary films only one diverticulum was visible; subsequent films plainly show other air-filled diverticula. Nielsen states that the acquired type often contains viscid mucus. Perhaps some of the diverticula were not originally visible because they were filled with mucus or were

collapsed. It is also interesting that this man had a nonproductive cough for approximately four years, which might have been a factor in the development of these lesions.

Ziegelman [13] states that these lesions may act as a reservoir for infection of the lower respiratory tract and/or disturb the recurrent laryngeal nerve. In addition, they are often associated with chronic respiratory disease. However, a review of the literature fails to disclose any record of medical or surgical treatment of tracheal diverticula. No treatment is contemplated in the case reported.

Summary

A case of tracheal diverticulosis is presented, with a brief review of the literature.

Drs. L. Henry Garland, Merrell A. Sisson, and Robert McNaught assisted in this work.

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REFERENCES

1. Rokitansky, K.: Handbuch der pathologischen Anatomie, Vienna, Braumüller & Seidel, 1846, Vol. 3, pp. 6, 7, and 11; Oesterr. med. Jahrb., 1838, Vol. 16.

2. Chiari, O.: Chirurgie des Kehlkopfes und der Luftröhre, Stuttgart, Ferdinand Enke, 1916, p. 217.

3. Ziegler and Kahlden: Über ein Pulsionsdivertikel der Trachea mit Bemerkungen über das Verhalten der elastischen Fasern an normalen Tracheen und Bronchien. Zentralbl. allg. Path. 8:721, 1897.

4. Kahler, O.: Über Divertikel des Tracheobronchialbaumes, Monatsschr. Ohrenh. 45:86, 1911.

5. McKinnon, D.: Tracheal Diverticula, J. Path. & Bact. 65:513, 1953.

6. Drymalski, G. W.; Thompson, J. R., and Sweany, H. C.: Tracheal Diverticula: Report of Case, Am. J. Roentgenol. 60:403, 1948.

7. Stibbe, E. P.: True Congenital Diverticulum of the Trachea in a Subject Showing also Right Aortic Arch, J. Anat. 64:62, 1929-1930.

8. Kahlstrom, S. C.: Tracheactasis, M. Bull. Vet. Admin. 18:218, 1943.

9. Nielsen, K.: A Case of Multiple Tracheal Diverticula, Acta radiol. 29:331, 1948.

10. Kernan, J. D.: Congenital Papilloma of Trachea—Diverticulum of the Trachea—Rupture of Diverticulum of Trachea into Innominate Artery, Ann. Otol. Rhin. & Laryng. 45:865, 1936.

11. Addington, E.; Rusk, P., and Cohen, W.: Tracheocele, Am. J. Roentgenol. 52:412, 1944.

12. Miller, W. S.: The Trachealis Muscle, Anat. Rec. 7:373, 1913.

13. Ziegelman, E. F.: Tracheal Diverticulum, Arch. Otolaryng. 21:414, 1935.

Development of the Inner Ear After Maternal Hypoxia

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The present investigation forms part of a systematic series of experiments designed to study the effect of maternal stress upon the unborn progeny,[8] the aim here being to appraise the effect of prenatal hypoxia upon the embryonic hearing organ. In the course of preceding investigations, stage-specific susceptibilities of different tissues to maternal hypoxia have been revealed, the nature of which is determined, among other factors, by sequences of development intrinsic in the embryo itself. Thus gross cerebral and skeletal defects followed critical hypoxic stresses on the 9th day, cleft palate those administered on the 15th day, "open eye" on the 16th and 17th days of pregnancy,[19] and retinovascular abnormalities in the neonatal period have been demonstrated microscopically.[7] The findings to date indicate that congenital defects stem from normal patterns of vascular development at the time when the impact of maternal stress registers upon the fetus. And in this respect, it is to be borne in mind that the embryonic inner ear is a relatively avascular tissue.

Materials and Methods

As in other experiments of the series, female white mice were chosen arbitrarily from the laboratory strain which has been maintained in vigorous health at this department for 10 years. For the past four years mating of parent to offspring or brother to sister has not been practiced in an effort to avoid inbreeding and thus simulate the incest taboos which determine the reproductive habits of human beings. The day on which the copulation plug appeared was regarded as the 1st day of pregnancy, and the uterine contents were delivered by surgical section on the 19th day.

The hypoxic atmosphere was created within a pressure chamber by a vacuum pump steadily exhausting the jar, while entrance of air into the system was restricted by means of an adjustable globe valve. By this device the degree and duration of hypoxia could be controlled and quantitated, while the continuous flow through the system of over 1 liter of air a minute prevented asphyxia, even of the low pressures used. A water bottle added to the circuit standardized humidity and temperature of incoming air, and the time for decompression and recompression was carefully regulated—at approximately 15 minutes.

The fetuses chosen for study of the hearing organ had undergone hypoxia indirectly through the maternal organism for five hours at low atmospheric pressures, as shown in Tables 1 and 2. Mothers were subjected to hypoxia on a single occasion between the 9th (8.5) and the 18th (17.5) day of gestation; and the simulated altitudes were, respectively, 25,000 ft. (280 mm. of Hg) for 2; 27,000 ft. (260 mm. of Hg) for 10; 35,000 ft. (178 mm. of Hg) for 5 animals, and 40,000 ft. (140 mm. of Hg) for 1 animal.

For investigations of the hearing organ, the head was easily severed from the body, a period

TABLE 1.—*Numbered Specimens Arranged by Day of Gestation on Which the Maternal Organism Was Exposed to Simulated Altitude*

Mouse	Day of Gestation	Pressure (mm. Hg)	Equivalent Altitude (ft.)
472	8.5	178	35,000
478	8.5	140	40,000
515	9.5	178	35,000
702	9.5	178	35,000
644	10.5	178	35,000
646	10.5	178	35,000
320	11.5	260	27,000
363	12.5	260	27,000
210	13.5	260	27,000
361	14.5	280	25,000
156	15.5	260	27,000
353	16.5	280	25,000
149	16.5	260	27,000
198	16.5	260	27,000
145	17.5	260	27,000
165	17.5	260	27,000
162	17.5	260	27,000
174	17.5	260	27,000
883 *			

* The mother of this animal had been exposed to approximately 10% oxygen at normal atmospheric pressure from day 7.5 of gestation to day 10.5 of gestation.

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From the Department of Epidemiology, Harvard University School of Public Health, and the Department of Otolaryngology, Harvard Medical School.

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TABLE 2.—*Numbered Specimens Arranged by Order of Simulated Altitudes to Which Maternal Organism Had Been Exposed*

25,000 ft.	27,000 ft.	35,000 ft.	40,000 ft.
353	145	472	478
361	149	515	
	156	644	
	162	646	
	165	702	
	174		
	188		
	210		
	320		
	363		

of fixation of 12 to 18 months having made decalcification unnecessary. After evacuation of the brain a segment was sliced from the cranial base containing both hearing organs in the same block. Some of the specimens were embedded in paraffin, others in celloidin. Sectioning followed in one of two planes: frontal or horizontal (parallel to the cranial base). Sections were 4 to 6μ thick for the paraffin-embedded blocks and 18 to 20μ for the celloidin blocks. Every 10th section of the series was stained with hematoxylin-eosin or one of several auxiliary stains. Twenty-four heads were prepared in this way, seven in frontal and seventeen in horizontal series, and examined in full and polarized light and under phase contrast.

The histological work was done by Miss Dorothy Linden; the photomicrograms were prepared by Mr. Donald C. Whitee.

Results

The results of these experiments were essentially negative. But the very paucity of findings is an indication for recording and discussing them. Findings are presented by age (length of gestation) at which the embryo experienced hypoxia; but, being negative, they can be considered essentially as if they illustrated the normal status of development after 18.5 days of gestation.

In an animal submitted to extreme maternal hypoxia (40,000 ft. of simulated altitude) on the ninth day of pregnancy extraordinary little is found considering the intensity and length of the hypoxic stress and considering that this is a period when skeletal anomalies are regularly produced by lesser stresses. A horizontal section cutting through both ears (Fig. 1) reveals the Eustachian tubes fully patent and free of hemorrhage, exudate, or detritus around their tympanic orifice with associated pneumatization of the middle ear. On the left, the stapes is shown with the stapedial artery intact and filling the lumen of the obturator foramen. The coils of the undifferentiated organ of Corti are seen in section, and endolymphatic parts of the vestibular labyrinth appear as narrow ducts compressed by perilymphatic edema.

In another animal (Fig. 2), that had been exposed on the same day of gestation to 35,000 ft. of simulated altitude, all three ossicles are discernible. However, they are still undifferentiated, avascular, and even on the 19th day of gestation are completely embedded in mesenchyma. The facial nerve lies above the stapes; and the vestibulum,

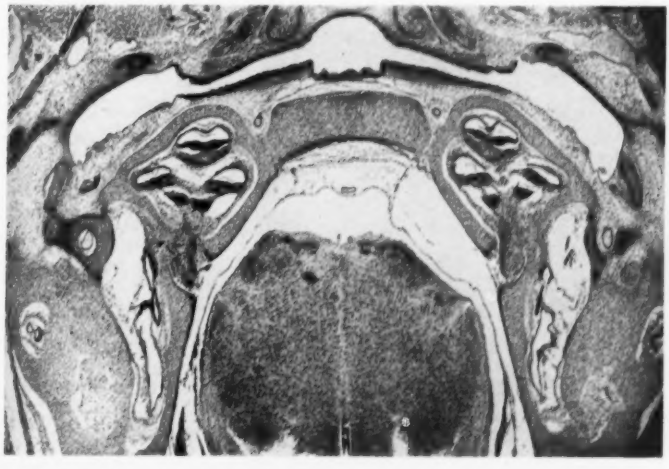

Fig. 1.—Horizontal section through both ears. Eustachian tubes wide open; around their tympanic orifice pneumatization of the middle ear starts. Reduced 1/3 from mag. $\times$ 37.

Fig. 2.—Cartilaginous ossicles. Middle ear filled by mesenchyma; utricular macula. Reduced 1/3 from mag. × 125.

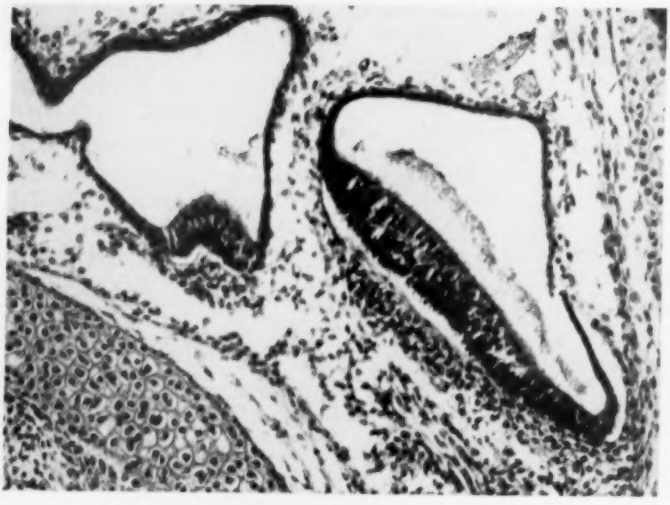

Fig. 3.—Utricular macula; horizontal crista. Endolymphatic space already free of mesenchyma; perilymphatic space still filled by mesenchyma. Reduced ¼ from mag. × 250.

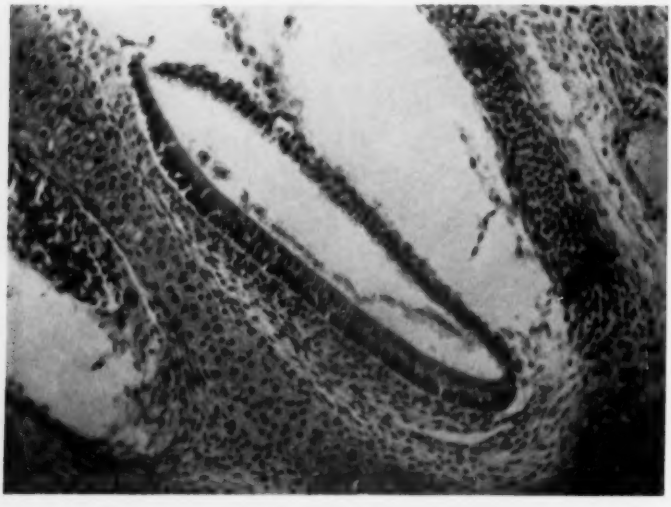

Fig. 4.—Edema in the perilymphatic space; saccule compressed; reduced ⅓ from mag. × 250.

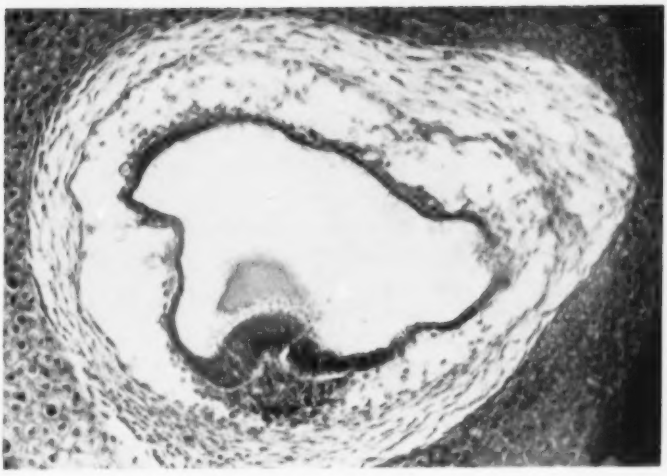

Fig. 5.—Posterior canal; layers of endosteum loosened; perilymphatic space distended by edema; reduced ⅓ from mag. × 250.

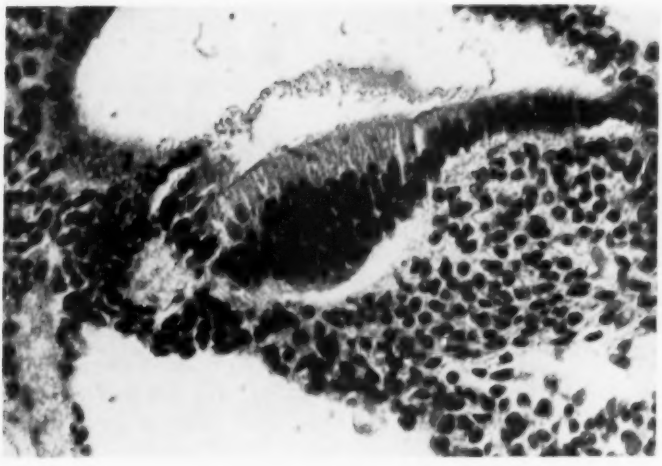

Fig. 6.—Cochlear duct; greater and lesser epithelial ridges; tectorial membrane poorly preserved but extending completely over both ridges; reduced ¼ from mag. × 450.

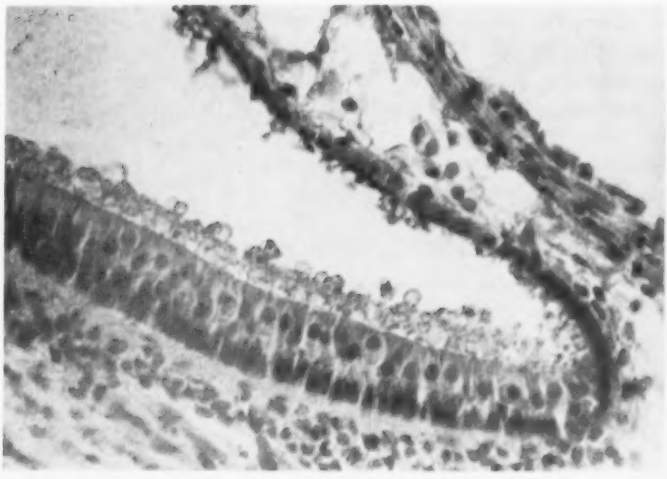

Fig. 7.—Saccular macula, with sensory hairs entering the layer of otolith crystals; reduced ⅓ from mag. × 400.

the perilymphatic cisterna, and the utriculus (seen in cross section) are practically free of mesenchyma. The macula of the utricle is supported, at its base, by a mesenchymal cushion, with its otolith layer detached.

High-power study of the utricular macula and the horizontal crista after exposure of the mother to an altitude of 35,000 ft. on the 11th day of gestation reveals those structures to be well on the way of development. Hair cells with their hair are seen; the otolith membrane is detached and mesenchyma fills the perilymphatic space, especially at the side where it supports the base of the macula. The endolymphatic space is already free of mesenchyma.

After exposure to 25,000 ft. on the 15th day of pregnancy the saccule (Fig. 4) is seen compressed by edema in the perilymphatic space, the latter being only partially free of mesenchyma. Hair cells with hair are distinguishable; the otolith membrane is partly detached. In the embryo shown in Figure 5, the contents of the cochlear duct have a strikingly healthy appearance considering that the mother was exposed at 25,000 ft. of the simulated altitude just two days before this embryo was removed surgically. The lumen of the duct is free of mesenchyma, and the tympanic scala is only partly formed by reabsorption of the mesenchyma. This was also the case for the vestibular scala, not shown in the photograph.

After exposure to 27,000 ft. on the 18th day (Fig. 6) the endolymphatic space is conspicuous in the posterior canal. The endosteum is loosely arranged, and the partly mesenchyma-filled perilymph space is dilated, compressing the endolymphatic canal with irregular contours. A crista with hair cells including hair is well formed, with the cupula shown in its proximal half.

It is to be borne in mind that the photographic illustrations shown depict development in animals that were killed about the 19th day of gestation. For comparison, the following illustrations reproduce the normal condition shortly after birth, which ordinarily normally occurs on the 20th day of pregnancy.

The saccular macula is shown fully formed in Figure 7, with hair entering the layer of otolith crystals. The endolymphatic space is free, with the perilymph still containing a considerable amount of mesenchyma.

In Figure 8, the ampullar cristae are formed in all detail, including the cupula with its striated structure.

The contents of the cochlear duct are shown in Figure 9 and, at higher power, in Figure 10. The sensory epithelium is still far from its mature form. Vestibular and tympanic scalae are partly filled with mesenchyma, from which only insignificant rests remain in the cochlear duct: This way the membrane of Reissner is covered by mesenchyma only at its aspect turned against the vestibular scala. The space of the internal sulcus is still occupied by the greater epithelial ridge (right side of Figs. 9 and 10), with the lesser epithelial ridge following laterally. Of the four hair cells which are clearly seen, the single inner one belongs to the greater epithelial ridge, forming its outermost edge, and the three externally placed belong to the lesser epithelial ridge. Internal and external hair cells are leaning against each other at an angle, as they are found later in their respective positions around the tunnel; the latter, however, is not yet formed. The tectorial membrane is firmly adherent to the surface of the epithelial ridges in all its length. In the basilar membrane, the vas spirale is present and marks the location of the base of the future tunnel.

Comment

Not even the severest hypoxic stresses to which these animals were exposed (namely, 35,000 or 40,000 ft. of simulated altitude of five hours' duration) induced recognizable deformity, either gross or microscopic, of the hearing organ. However, enough evidence has been assembled about the pathogenesis of defects elsewhere in the body—

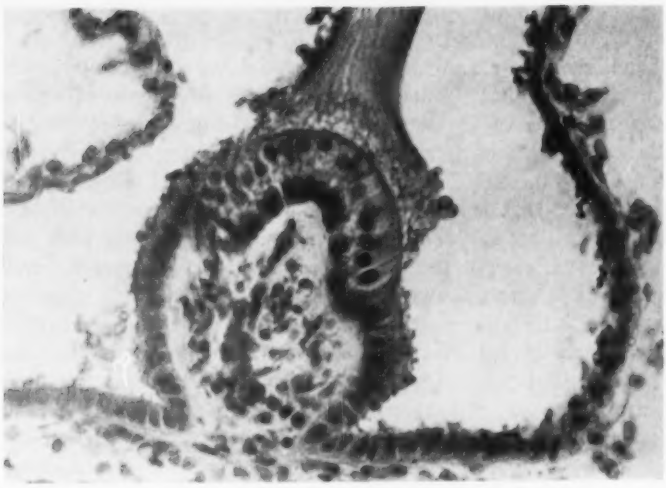

Fig. 8.—Ampullar crista with cupula; reduced ⅓ from mag. × 400.

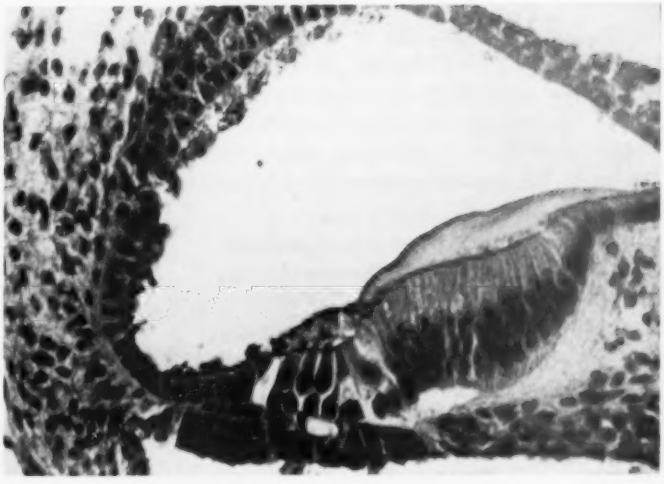

Fig. 9.—Contents of the cochlear duct. Greater epithelial ridge at right; lesser epithelial ridge at left. Tectorial membrane adherent. Reduced ⅓ from mag. × 400.

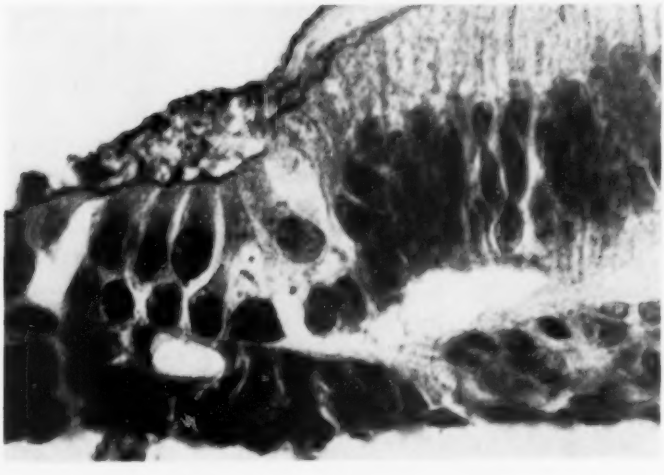

Fig. 10.—High-power view of part of Figure 9; reduced ⅓ from mag. × 1000.

the brain, palate, eyes, and skeleton—to allow a reasonable interpretation of these negative findings to be advanced. The very fact that the deformities produced by hypoxia to date have been mediated through the vascular system and have occurred at sites undergoing spurts of capillary growth makes it intelligible that the inner ear should withstand disturbances capable of producing structural anomalies elsewhere, for the embryonic ear, on the days when the maternal-fetal organisms were being stressed, at least, is a relatively avascular organ. A principle governing the development of congenital anomalies seems to be that resting structures, i. e., primordia and undifferentiated precursors like the ossicular chain shown in Figure 2 as well as fully differentiated organs, are relatively resistant to teratological injury by comparison with the tissue undergoing rapid vascular expansion and differentiation at the same time—the fundus of the eye, for example, during the second week after birth.[7]

It is difficult to evaluate the significance of what might be perilymphatic edema in some of the sections described, whether it was real or an artifact. Detachment of the soft parts from the rigid bony or cartilaginous walls in the ear—and similarly in the nasal cavity—is always difficult to evaluate, for these soft-tissue linings have a tendency to shrink, especially after fixation in alcohol. Histologically they then appear taut and stretched out in the shortest line between points of suspension. This gives them the appearance of being detached from the wall. In the vestibular labyrinth compression of the endolymphatic channels can be produced in this manner. However, shrinkage could hardly produce such loosening of the endosteal cell layer as is revealed in Figure 6. Such changes and some of the perilymphatic dilatation, with resultant compression of the endolymphatic space, cannot be disregarded and may represent functional response to what, after all, is close to being an agonal stress.

It is of doubtful value to compare the studies reported here with those of others who have been studying the effect of hypoxia on the adult hearing organ. However, as early as 1882, Kovács and Kertész [14] produced nystagmus in the rabbit and recorded changes in severity with different degrees of anoxia. Werner [21] found that the cells of the macula sacculi in guinea pigs, but not those of the macula utriculi, increase in height with higher and decrease in height with lower atmospheric pressures. Aschan [1] gave 9 to 10 volume per cent oxygen to rats and rabbits for six-hour periods on 10 consecutive days. Inflammatory changes were more pronounced in his test animals than in the controls, and in the middle ear, edema, hyperemia, and extravasations of red blood cells and plasma were present.

A comparison with present findings is hardly possible, as in the mouse embryo pneumatization of the middle ear is just starting. More pertinent is the finding of Marullo [16] that an atmospheric hypotension of 200-500 mm. of Hg had no malformative effect on aural development in chick embryos. And Kelemen [9] was unable to find abnormalities of aural development in the young after radiation damage of the pregnant rat.

It is possibly of interest that Lawrence and Wever [15] had to expose cats to a mixture of nitrogen and air with an oxygen content of only 0.5% in order to induce disintegration of the elements of the organ of Corti; and, in 1949, Wever, Lawrence, Hemphill, and Straut [22] postulated from experiments on cats that it was necessary to breathe less than 4% oxygen—a partial pressure corresponding to an altitude exceeding 40,000 ft. above sea level—to obtain deleterious effects on the ear. The implications are those of relative resistance to hypoxia of the organ of Corti in comparison with more susceptible soft tissues—like the brain—and rapidly differentiating organs. Likewise, there has been considerable experimental work done by Bornschein,[2,3]

Gisselson,[6] Fernandez,[4] Tonndorf,[18] and their colleagues on the effect of hypoxia on cochlear potentials, and Gellhorn [5] has studied the sensitivity of the auditory projection area to anoxia in cats. However, its bearing on the present investigation is too problematical for discussion.

Regarding cochlear potentials, however, the development of the hair cells toward the end of gestation in the mouse suggests that these cells might be selectively injured in prenatal life. In any case, the evidence presented here indicates a timetable of prenatal development not previously identified, although the sequences between birth and maturation have been worked out by Wada.[20]

In the over-all series of laboratory investigations many kinds of anomaly have been induced in organs other than the ear; they include cranioschisis, hemivertebra, rib defects, kinky tail, cleft palate, and variations of open eye and microphthalmia. Congenital vascular defects also were observed in the skin, in the subcutaneous tissue, the eye, and the central nervous system. These vessel changes corresponded to a gradient represented by related processes such as vascular nevi, telangiectasias, and cavernous lymphangiomas. It is possible that the perilymphatic edema as observed in the vestibular labyrinth is to be fitted into this framework, but such an interpretation would be purely speculative.

After complications of pregnancy in human beings, such as rubella (Schall, Lurie, and Kelemen [17]), diabetes, poliomyelitis, erythroblastosis fetalis, and nephritis (Kelemen [10-13]), the status of the hearing organ in the newborn was found comparable to that reported here. Sensory end-organs were found to be essentially intact, and whatever lesions were observed belonged in the realm of vascular pathology. In regard to vascular sequences, however, the roles of prematurity were stressed and anoxia was interpreted as an important factor.

Summary

Pregnant mice were exposed to anoxia of five hours' duration at different stages—between the 9th and the 18th day of pregnancy. The embryos were removed surgically about a day before term, and the inner ears were examined under the microscope.

The sensory end-organs of the inner ear were found intact and in adequate developmental stage. No significant differences were noted among different specimens no matter what the gestational day on which hypoxic injury was inflicted or what the simulated altitude between 25,000 and 40,000 ft.

Perilymphatic edema associated with compression of the endolymphatic tube was seemingly present with greater than expected frequency. This finding, however, needs further scrutiny in the light of histotechnical considerations suggesting that it could be an artifact of fixation.

The negative findings are the more striking considering that anoxic damage to the vascular and nervous systems was readily demonstrable in many of the same animals. Probably the independent development of the end-organs from the nervous system and the primitive state of their vascularization at the periods selected for study contributed to their intact development.

A similar relative resistance of the sensory organs of the inner ear has been found in animals exposed to radiation injury and in human embryonic studies of otic development after maternal rubella, erythroblastosis, diabetes, and nephritis.

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REFERENCES

1. Aschan, G. K.: Aero-Otitis Media and Aerosinusitis, Acta oto-laryng., Supp. 69, pp. 1-93, 1948.

2. Bornschein, H., and Krejci, F.: Das Verhalten der Cochlearpotentiale bei Sauerstoffmangel, Monatsschr. Ohrenh. 83:190-196 (April-June) 1949.

3. Bornschein, H., and Krejci, F.: Untersuchungen über die Kreislaufabhängigkeit der Cochlearpotentiale bei Anoxie, Monatsschr. Ohrenh. 83:386-392 (Oct.-Dec.) 1949.

4. Fernandez, C.: The Effect of Oxygen Lack on Cochlear Potentials, Ann. Otol. Rhin. & Laryng. 64:1193-1203 (Dec.) 1955.

5. Gellhorn, E.: Sensitivity of the Auditory Projection Area to Anoxia, Am. J. Physiol. 164: 748-751 (March) 1951.

6. Gisselson, L.: The Effect of Oxygen Lack and Decreased Blood Pressure on the Microphonic Response of the Cochlea, Acta oto-laryng. 44:101-118 (March) 1954.

7. Habegger, H.; Philbrook, F. R.; Curley, F. J., and Ingalls, T. H.: Retinopathy of Immaturity: In the Newborn Mouse After Exposure to Oxygen Imbalances, Am. J. Ophth. 42:377-392 (Sept.) 1956.

8. Ingalls, T. H.; Curley, F. J., and Prindle, R. A.: Experimental Production of Congenital Anomalies: Timing and Degree of Anoxia as Factors Causing Fetal Deaths and Congenital Anomalies in the Mouse, New England J. Med. 247:758-768 (Nov. 13) 1952.

9. Kelemen, G.: Experimental Defects in the Ear and the Upper Airways Induced by Radiation, A. M. A. Arch. Otolaryng. 61:405-418 (April) 1955.

10. Kelemen, G.: Aural Changes in the Embryo of a Diabetic Mother, A. M. A. Arch. Otolaryng. 62:357-369 (Oct.) 1955.

11. Kelemen, G.: Acute Poliomyelitis of the Mother, with Aural Lesions in the Premature Infant, A. M. A. Arch. Otolaryng. 62:602-610 (Dec.) 1955.

12. Kelemen, G.: Erythroblastosis Fetalis: Pathologic Report on the Hearing Organs of a Newborn Infant, A. M. A. Arch. Otolaryng. 63: 392-398 (April) 1956.

13. Kelemen, G.: Embryonaler Ohrbefund nach operativer Entfernung wegen mütterlicher Nephritis, to be published.

14. Kovács, L., and Kertész, J.: Über die Wirkung einiger chemischen Stoffe auf die associierten Augenbewegungen, Arch. exper. Path. u. Pharmakol. 16:81-104 (July) 1882.

15. Lawrence, M., and Wever, E. G.: Effects of Oxygen Deprivation upon the Structure of the Organ of Corti, A. M. A. Arch. Otolaryng. 55: 31-37 (Jan.) 1952.

16. Marullo, T.: Dell'influenza di ipotensioni atmosferiche sulla organogenesi dell'orecchio del pulcino, Clin. otorinolaring. 7:276-291 (June) 1955.

17. Schall, L. A.; Lurie, M. H., and Kelemen, G.: Embryonic Hearing Organs After Maternal Rubella, Laryngoscope 61:99-112 (Feb.) 1951.

18. Tonndorf, J.; Hyde, R. W., and Brogan, F. A.: Combined Effect of Sound and Oxygen Deprivation upon Cochlear Microphonics in Guinea Pigs, Ann. Otol. Rhin. & Laryng. 64: 392-405 (June) 1955.

19. Tedeschi, C. G., and Ingalls, T. H.: Vascular Anomalies of Mouse Fetuses Exposed to Anoxia During Pregnancy, Am. J. Obst. & Gynec. 71:16-28 (Jan.) 1956.

20. Wada, T.: Anatomical and Physiological Studies on the Growth of the Inner Ear of the Albino Rat, Memoir 10, Philadelphia, Wistar Institute of Anatomy and Biology, 1923.

21. Werner, C. F.: Zellreaktionen im inneren Ohr bei Veränderungen des Luftdruckes, Ztschr. Hals- Nasen- u. Ohrenh. 32:544-549 (March) 1933.

22. Wever, E. G.; Lawrence, M.; Hemphill, R. W., and Straut, C. B.: Effects of Oxygen Deprivation upon the Cochlear Potentials, Am. J. Physiol. 159:199-208 (Nov.) 1949.

Modern Treatment of Sinusitis

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The treatment of sinusitis has undergone remarkable changes as a result of twin impacts. One is the management of infection by using antibiotics; the other is the control of allergic manifestations. In addition to, and in part because of, these concepts, advances have been made in surgical techniques, but surgical procedures have become statistically less frequent and will be considered only incidentally.

The emphasis in diagnosis has changed. Although we still need to find out which sinuses are involved, we particularly need information regarding the following points:

1. The infecting bacteria, identified as to its sensitivity to antibiotics.
2. The allergic status of the patient.
3. The general medical condition, including temperature, fatigue and stress, nutrition and blood reactions, and impaired thyroid function and other endocrine abnormalities.
4. The cytologic pathology, which provides evidence of viral or bacterial activity and allergic reaction.

If, as so often happens, it is a case of sinusitis arising from a neglected upper respiratory infection, which is neither chronic nor recurrent, and is of mild or moderate severity, a tentative working diagnosis is usually sufficient for starting treatment. I am sure that we all find that most of these cases respond promptly to a broad-spectrum antibiotic, along with local and systemic vasoconstrictors.

The old need for drainage of infected sinus cavities is still as great as ever, but our medical means of accomplishing it have become more effective and have displaced to a considerable extent the surgical methods of attaining it. Until almost 1930, cocaine and epinephrine were about the only vasoconstrictors. Antihistamines were unknown, and control or even diagnosis of allergic edema was rarely attained. Methods of irrigation were limited to the nose, as such, or by trocar. The Proetz [13] displacement method for treatment had yet to be described. But now with our increasing armamentarium of drugs and with the Proetz displacement method being a routine procedure in most offices, many acute cases respond in short periods of time and stay well.

We still, however, have the neglected cases and in addition, from time to time, we see unusually severe acute infections as well as recurrent and chronic cases. Antibiotics may have been used repeatedly along with local treatments. Operative procedures may have been carried out, but the patient remains sick. These cases constitute our main problems in handling sinus disease.

When the history reveals chronic or recurrent nasal trouble, with or without a present acute episode, the most complete study of the case that is possible should be made. The history needs to be expanded in an attempt to uncover other possible causes, namely, allergic, metabolic, hygienic, and nutritional. In order to be complete and to save time, Anderson [1] devised and published a detailed questionnaire. If allergy is suspected, the questioning is directed to determine correlations between symptoms and environment. The dental status should not be overlooked.

The examination has not changed much in the past 30 years, but we have a few important additions to it. One of these is a careful microscopic study of the cellular pathology. The presence of eosinophiles in

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nasal secretions is widely accepted as an indication of allergic response. Hansel [7] has stressed for years the importance of the eosinophilic-neutrophilic ratio in cytology for interpreting the interplay of allergic response and infection. Hilding [8] as early as 1930 showed that during the common cold the columnar ciliated epithelial cells of the nose were exfoliated and were seen in the nasal secretions from these patients. In 1950, by use of the Papanicolaou stain, we [4] were able to show specific patterns of structural changes that occurred within the ciliated cells during acute upper respiratory infections. These intracellular changes have proved to be of diagnostic value in differentiating the common cold from allergic response in the nose. Since the ciliated epithelial cells are involved in nasal disease, an additional factor facilitating cytological observations of the condition of these cells as well as the other cells present in the secretions has been found in our use of the "wiped nasal smear." It is really a painless nontraumatic biopsy of the nose, and it has been my practice for three of four years to make these smears on all problem cases. In allergic noses the columnar epithelial cells are not as frequently exfoliated as they are in the common cold, but with the wiped smear they are easily obtained for study and are representative of the condition of the mucosa. The allergic edema actually can be seen within the individual columnar cells. Goblet-cell formation is noted, and this finding seems to be more constantly present in allergic noses than the finding of eosinophiles. The information obtained thereby is helpful in establishing differential diagnosis.

In making the wiped smear I pass a small cotton-tipped applicator under the inferior turbinate, make the smear before it has time to dry, and place the slide in equal parts of 95% alcohol and ether for fixation. Drying in the air or flaming the slide will destroy much of cellular detail. Two smears are made so that two different stains can be used: Wright's or Hansel's stain, for the granulocytes and mononuclears, and Papanicolaou's stain, which is especially good for demonstrating epithelial cells. (We feel that the complete cytological picture is more reliable and informative.)

Since we are rarely justified in taking even minute biopsy specimens of the nasal mucosa unless tumor is suspected, this simple method of the "wiped nasal smear" furnishes us with direct evidence of allergic response, and viral and bacterial infection, even when they are present simultaneously. If tumor cells happen to be present they are readily detected in the same preparation and give the warning for the necessity of regular biopsy.

The bacterial culture has taken on new practical values with the introduction of the numerous antibiotics. The selection of which antibiotic to use can be guided by sensitivity tests. This has become increasingly important, since bacteria have been gaining resistance to frequently used antibiotics.[5] Response to an antibiotic is usually prompt—within 48 hours; so if the first antibiotic has not started improvement in that time, a change is in order even without laboratory reports.

When the acute process is under control with temperature and tenderness subsiding, more definite local treatment is indicated. Before the use of antibiotics this period rarely occurred before two weeks of acute infection. At this time during the subacute stage, I find that I rely mainly upon the Proetz displacement treatment and upon antrum irrigations for local therapy, as is shown in Table 1. These 111 cases were found in a group of 1500 consecutive case records from my files of 1953-1955.

TABLE 1.—*Treatments Given to 111 Cases of Sinusitis*

Type of Treatment	No. of Cases
Displacements	59
Irrigation of antrum	27
Both D. and I.	10
D. and I. and	
Caldwell-Luc	2
Polyethylene tube	1
Polypectomy	1
Subtotal	100
Medical treatments only	11
Total	111

Fig. 1.—Normal ciliated epithelial cells showing oval nuclei with nucleoli, finely granular chromatin structure, and homogeneous cytoplasm. The cilia and ciliary bodies are prominent.

Fig. 2.—Ciliated cell showing slightly more advanced stage of degeneration. The cytoplasm is coarsely granular, and the chromatin masses are larger. The pink intranuclear inclusion body is prominent.

Fig. 3.—Advanced stage of degeneration in a ciliated cell with completely pyknotic nucleus, halo, perinuclear bodies, and large pink globule in cytoplasm. Some cilia appear on the side of the cell.

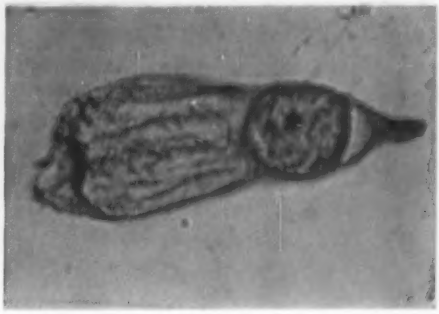

Fig. 4.—Columnar epithelial cell wiped from an allergic nose which shows marked edema and vacuolization of the cytoplasm. The nuclear membrane is thickened, outside of which on one side is a large vacuole. The cilia are degenerated.

TABLE 2.—*Irrigation in Seventeen Cases of Bilateral Chronic Antral Infection*

		Number of Irrigations per Antrum Until Clear																
Sulfonamide	(Rt)	2	2	3	3	3	3	3	3	4	4	4	6	6	7	7	7	8
Saline	(Lft)	2	2*	2	3	4*	5	6*	8	4	5	6	7	9	5	8*	9	8

		Antra Slower to Clear	Total Irrigations
Sulfonamide	(Rt)	2	75
Saline	(Lft)	11	93

* These four antra were not cleared.

After seeing Pomerat's [12] pictures on the devastating effect of isotonic saline on the tissue cells growing in cultures, I changed to Ringer's solution for irrigations in these treatments. I usually add to the solution a vasoconstrictor, an antihistamine, and antibiotics (not suitable for systemic use). The rationale of the first two is in keeping with the need to obtain drainage by decreasing congestion, and patients seem to like it.

The local use of antibiotics may be debated. In 1945 we tabulated all antrum irrigations in the clinic (Washington University). Cultures were made. The bacteria most frequently found were alpha Streptococcus, Hemophilus influenzae, Neisseria catarrhalis, and pneumococci. In the chronic cases several kinds of bacteria were usually present. Some of the data are presented in Table 2.

All right antra after irrigation had instilled a solution of vasoconstrictor and sulfathiazole or sulfadiazine, while all left antra were irrigated and had no sulfonamide instillation. The control is considered to be the left side of each pair of antra. The number is unfortunately small. There were another 20 cases that stopped treatment before clearing. A tendency is noted for the sulfonamide-treated side to become clear before the other side. There was a total of 75 irrigations when a sulfonamide was instilled compared with 93 when it was not used, and in the pairs two were worse with a sulfonamide, whereas 11 were worse without it. Theoretically it is necessary for the antibiotic to be in contact with the bacteria for a considerable time. A higher concentration can be used in irrigating solutions than is obtained parenterally. If the solution does not run out too fast it may help more than parenteral medication. Later a suspension of sulfathiazole was used. However, if this is used too frequently it can cause foreign-body formation. (I saw one case with the antrum packed with rock-hard sulfathiazole as it was being removed via a Caldwell-Luc operation.)

We now have better agents, and, having found by culture and sensitivity test which antibiotics are effective, we can safely use them locally even if such antibiotics are unsuitable for systemic use. I tend to use locally those which are unsuitable for systemic use, since there is less chance of sensitizing the patient to an important medication. In stubborn cases I have instilled the antibiotic in a suspension or ointment, without harm. I do not omit giving the antibiotic systemically, but the concentration of antibiotic can be so much higher if the systemic use is augmented with local use.

The unusual antrum that fails to respond to the above measures may be treated by means of an indwelling plastic catheter, as recommended by Klaff.[10] Polyps, benign tumors, marked hyperplasia of the mucosal lining, and structural abnormalities occur and hinder healing. These may have to be corrected before recovery can be obtained.

Occasionally frontal sinuses do not respond to general management. The frontal sinus is more difficult to drain owing to its long devious duct. In the rare fulminating acute case with beginning intracranial extension, drainage may safely be obtained through a trephine opening in its floor (orbital wall), as advocated by J. McKenzie Brown.[3] I have used it also in subacute cases, with satisfactory results. Adequate drainage is afforded, and local instillations will augment systemic antibiotic treatment.

It has been many years since I have had to catheterize and irrigate a sphenoid sinus.

No doubt such cases occasionally occur, but most sphenoids and ethmoids respond well to medical treatment and displacements.[16]

There are some cases of persistent chronic sinusitis with or due to allergy which are not to be confused with the many cases of allergic rhinitis which are called "chronic sinus" by laymen. Recent articles[3,11,16,17] all agree on the importance of allergic management in treating these cases. Allergy is a demonstrable and treatable factor in some cases of weakened resistance. It is also a cause of obstructive edema. If allergic factors have been detected they should be controlled. Antihistamines have been valuable. For definitive allergic work-up, I follow a plan closely based upon that of Rinkel.[14] In this plan, to start treatment, inhalants are quantitatively tested by serial dilutions (that is, titration), and dosage is later changed according to the response. Food allergy may be quite obscure. It is surprisingly overlooked, even when the case is worked up from every other angle. Foods are diagnosed by individual food tests and analytic diet record. The plan is clear-cut, but the mass of detail is formidable.

Vaccines have been used by some for many years (St. Clair Thompson). I occasionally use them in three types of cases:

1. Cases with frequent acute infections without detectable defects.

2. Cases treated with prolonged and repeated courses of antibiotics.

3. Cases in which there is bacterial hypersensitivity or in which allergic symptoms seem due to bacteria.

TABLE 3.—*Treatments Given to 111 Cases of Sinusitis*

Type of Treatment	With Antibiotics	Allergy	Both	Neither	Totals
Displacements	7	13	9	30	59
Antrum irrigations	3	4	3	17	27
Both D. and I.	3	3	2	2	10
D. and I. and Polyethylene tube			1	..	1
Caldwell-Luc			1	..	1
Caldwell-Luc and submucous res.			1	..	1
Polypectomy			1	..	1
Subtotal					100
Medical only	5		6	..	11
Totals	18	20	24	49	111

Admittedly I feel that the use of vaccine is almost experimental; however, experience with extracts of fungi has been widespread and generally quite satisfactory. At present I am using bacterial vaccines in a similar manner with doses usually intradermally and small enough to avoid either marked local or systemic reactions.

Alevaire* and corticosteroids are used by some, but I have had too little experience to comment on their effects.

The frequency of medical treatment in cases of sinusitis is shown in Table 3. All of the cases had local medication to relieve congestion. Other medical treatment consisted primarily in antibiotics and allergic management. It is seen that 10% had medical treatment only. About half of the cases treated with displacements or antral irrigations had no systemic antibiotics or allergic treatments.

Summary

The importance of the medical treatment of sinusitis is stressed. In order to manage sinusitis most effectively one must have accurate diagnosis of the etiological factor, which include the sensitivity of the bacteria to the antibiotic, the allergic status, the endocrine function, and general medical information. The "wiped nasal smear" offers objective cytological evidence in regard to the condition of the nasal mucous membrane. Most cases can be cured when these factors are attacked simultaneously and when concomitant appropriate drainage of blocked or filled sinuses is employed. The surgical correction of structural impediments may also be necessary.

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* Alevaire is an aqueous solution of oxyethylated tertiary octylphenolformaldehyde polymer, 0.125%, in combination with 5% glycerin and 2% sodium bicarbonate.

REFERENCES

1. Anderson, J. R., and Rubin, W.: Guide for Use of the Allergy History Questionnaire and Summary Sheet, distributed by the Hansel Foundation, 634 N. Grand Ave., St. Louis.

2. Coates, G. M.; Schenck, H. P., and Miller, M. V., Editors: Otolaryngology, Hagerstown, Md., W. F. Prior Company, Inc., 1955, Vol. 3.

3. Brown, M. J., and Goodhill, V.: Maxillary and Frontal Sinusitis, in Otolaryngology,[2] Chap. 9.

4. Bryan, W. T. K., and Bryan, M. P.: Structural Changes in the Ciliated Epithelial Cells During the Common Cold, Tr. Am. Acad. Ophth. 57:297-303 (May-June) 1953.

5. Davison, F. W.: Antibiotics and Otolaryngologic Infections, Bull. Geisinger Memorial Hosp. & Foss Clin. 6: (Nov.) 1954.

6. Goldman, J. L.: Bacteriologic and Cytologic Criteria for Diagnosis in Nasal and Sinus Disease: Basis and Interpretation, Tr. Am. Acad. Ophth. 58:68-72 (Jan.-Feb.) 1954.

7. Hansel, F. K.: Clinical Allergy, St. Louis, C. V. Mosby Company, 1955.

8. Hilding, A. C.: The Common Cold, Arch. Otolaryng. 12:133-150 (Aug.) 1930.

9. Hill, F. T.: Symposium: Modern Treatment of Maxillary Sinusitis; Diagnosis of Chronic Maxillary Sinusitis; Indications for Surgery, Tr. Am. Acad. Ophth. 57:184-186 (March-April) 1953.

10. Klaff, D.: Personal communication to the author.

11. Lierle, D. M.: Sinusitis in Children, in Otolaryngology,[2] Chap. 11.

12. Pomerat, C. M.: Recent Observations on Living Human Cells with Cinematographic Technique, read before the Fifty-Ninth Annual Meeting of the American Laryngological, Rhinological and Otological Society, March 16, 1956 (not published).

13. Proetz, A. W.: Applied Physiology of the Nose, St. Louis, Annals Publishing Co., 1948.

14. Rinkel, H.: Food Allergy, Springfield, Ill., Charles C Thomas, Publisher, 1951.

15. Semenov, H.: The Pathology of the Nose and Paranasal Sinuses in Relation to Allergy, with Comments on Local Injection of Cortisone, Tr. Am. Acad. Ophth. 56:121-170 (March-April) 1952.

16. Smith, A. T.: Acute and Chronic Sphenoid and Ethmoid Sinus Diseases, in Otolaryngology,[2] Chap. 10.

17. Van Alyea, O. E.: Symposium: Modern Treatment of Maxillary Sinusitis; Nonsurgical Treatment of Acute and Chronic Maxillary Sinusitis, Tr. Am. Acad. Ophth. 57:180-183 (March-April) 1953.

Evaluation of Postlaryngectomy Rehabilitation Programs

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Clinical studies dealing with the psychological reaction of the patient to laryngectomy, and those studies which evaluate the progress made by laryngectomized patients in programs for speech rehabilitation, should include all patients referred to a given program. The significantly high percentage of patients who refuse to participate and those who, after a short period of participation, discontinue their cooperation and efforts are often ignored in reported clinical studies, and only "successful cases" are reported.

It is possible that in such studies some conclusions reached without considering this group would be misleading. The patient's personality structure and the physiological limitations which may be present are possibly of much importance in determining the degree of success which a given patient may have in the rehabilitation program. The reactions of the nonconforming or noncooperating group—per se—are important to a clinical study, and may well prove valuable to those who are trying to rehabilitate the largest possible number of patients. The error in disregarding the noncooperative group would be comparable to a research project on alcoholism in which only members of Alcoholics Anonymous were included.

Several examples from the literature are cited:

Pitkin,[1] in his detailed study of the patients' adjustments to the laryngectomy, sent questionnaires to approximately 200 patients. Of these, only 61 replied and their reactions were reported. Heaver, White, and Goldstein,[2] reporting on 274 laryngectomized patients admitted to The National Hospital for Speech Disorders during a two-year period, 1952-1953, state that 110 (40.1%) patients discontinued treatment against advice. They state: "Although one-third of this group had to cease their attendance because of such factors as the need for additional surgery, medical or serious emotional complications, generalized weakness, travel distance or demise, the remaining two-thirds apparently left the clinic because of overwhelming feelings of futility regarding the possibility of ever speaking as adequately as they once did. Their subjective estimate of what constituted improvement was differently based than the objective estimate of the therapist . . . . Because of postoperative physiologic disability or fixed emotional attitudes, 12 others (4.4%) ultimately found it necessary to use a mechanical aid—the electro-larynx."

It is significant to note that since the period covered in the report above, and in current practice by the postlaryngectomy speech department of The National Hospital for Speech Disorders, "the instrument is now routinely demonstrated to the new patient by the therapist at the time of his first therapeutic session . . . . He is told that he may use the electro-larynx immediately, if he chooses—also, if he prefers, he may have esophageal voice therapy for four to six weeks, without the instrument, to establish a fair trial to determine his capacity to learn and use the new voice; or, finally, if his economic status and job are in immediate jeopardy, he may concurrently take voice lessons and use the instrument while at work . . . . Whatever choice is made is entirely up to the patient."*

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* Communication from Lynwood Heaver, M.D., Director of The National Hospital for Speech Disorders, New York, Dec. 3, 1956.

Andrews and Kucera,[3] in reporting their observations of 71 patients with laryngeal carcinoma state: "The development of buccoesophageal voice after laryngectomy has been variable; the most important factor appears to be the motivation by the patient."

Figi and Divine,[4] and Greene,[5] discuss the laryngectomized patients' psychic trauma, and Levin[6] suggests several physical and psychological conditions which may be characteristic of those patients found in the noncooperating category, such as stenosis of the esophagus; resection of the cervical portion of the esophagus; multiple lesions, such as broncheal carcinoma in addition to the laryngeal lesion; dysfunction of the respiratory or cardiovascular system; loss of hearing, senility, or other feebleness.

In their studies of the breathing and speech coordinations of laryngectomized patients, Di Carlo, Amster, and Herer[7] suggest that many patients fail in their efforts to acquire an esophageal voice because they have not first been taught speech-breathing coordinations and voice control.

An examination of these studies reveals a high percentage of patients are not responsive to available rehabilitation programs. It is perhaps to these patients that Pitkin[1] refers when he says: " . . . the facts, as set forth by the patients, along with their comments and suggestions, indicate that there are many ways in which otolaryngologists are not fulfilling their responsibility toward these patients and that numerous practices might be improved to add to the patients' welfare and peace of mind. Responsibility devolves on the laryngologic surgeon when he carries out the procedure of separating the individual from his normal means of communication, and he—the surgeon—should follow the patient routinely through all the phases of preoperative preparation and postoperative care, so that he may be on the alert to detect any evidence of recurrence, become cognizant of the patient's emotional and psychologic difficulties, and contribute all in his power toward alleviating them."

It is suggested that a research project aimed toward an analysis of factors preventing cooperation by this group of patients might indicate a course of psychiatric therapy or prespeech training designed to prepare the patient for further rehabilitation, or, in some cases, a preference might be indicated for the prescription of a prosthesis for voice, such as the electrolarynx.

Contrary to Seemann's[8] conclusion in 1926 that "the artificial larynx belongs to the past," more recent papers by laryngologists and factual data from commercial sources show an increase in the prescription and use of the prosthesis. It is true that an improved prosthesis has been made available to the patient since 1926. It is also a fact that the incidence of laryngectomy has increased many times since that time, and, according to Martin,[9] it is probable that the incidence will continue to increase.

The laryngectomy patient seeking a substitute voice would naturally choose an effective, normal-sounding, socially adequate esophageal or pharyngeal voice in preference to the use of any prosthesis for speech, despite the relatively speedier and simpler learning process with the prostheses.

Hyman's[10] findings in connection with a preference expressed by auditors of the several types of speech, and the reactions of viewers to the cosmetic aspect of the different kinds of speakers, are not discussed herein, because the relative merits of postlaryngectomy speech modes are not germane to the immediate subject. Rather, the aim here is to seek a means for the rehabilitation of a greater proportion of laryngectomy patients than is now participating in rehabilitation programs and to provide them with a means of oral communication.

A team comprised of the laryngologist, the speech pathologist or esophageal voice teacher, and the psychologist would appear to be best fitted for the task of evaluating the factors inherent in the noncooperating group of patients. Broadly, the purpose of this team would be to determine, by physical and psychological examination, and by clini-

cal experience, whether the patient should be referred to psychiatric direction, prespeech training, or both, to help him achieve the maximum potentialities of a rehabilitation program, and whether esophageal voice training or a speech prosthesis is indicated to attain the maximum rehabilitation in his case.

Abandonment of the patient to his own subjective evaluation of his rehabilitation potentialities without a thorough investigation and effort to bring about the consummation of his true rehabilitation potentials seems remiss, and not compatible with the best tradition of medicine.

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REFERENCES

1. Pitkin, Y. N.: Factors Affecting Psychologic Adjustment in a Laryngectomized Patient, A. M. A. Arch. Otolaryng. 58:38-49 (July) 1953.

2. Heaver, L.; White, W., and Goldstein, N.: Clinical Experience in Restoring Oral Communication to 274 Laryngectomized Patients by Esophageal Voice, J. Am. Geriatrics Soc. 3:687-690 (Sept.) 1955.

3. Andrews, A. H., and Kucera, W.: Management of Laryngeal Carcinoma, A. M. A. Arch. Otolaryng. 57:108-109 (Jan.) 1953.

4. Figi, F. A., and Devine, K. D.: Tumors of the Nose and Throat, A. M. A. Arch. Otolaryng. 61:74-118 (Jan.) 1955.

5. Greene, J. S.: Laryngectomy and Its Psychologic Implications, New York J. Med. 47:53-56 (Jan. 1) 1947.

6. Levin, N. M.: Speech Rehabilitation After Total Removal of Larynx, J. A. M. A. 149:1281-1286 (Aug. 2) 1952.

7. Di Carlo, L. M.; Amster, W. W., and Herer, G. R.: Speech After Laryngectomy, Syracuse University Special Education and Rehabilitation Monograph Series 1, Syracuse, N. Y., Syracuse University Press, 1955.

8. Seemann, M.: Phoniatrische Bermerkungen zur Laryngektomie, Arch. klin. Chir. 140:285-298, 1926.

9. Martin, H.: The Incidence of Total and Partial Laryngectomy, 1947 and 1952, Cancer 8:1122-1125 (Nov.-Dec.) 1955.

10. Hyman, M.: An Experimental Study of Artificial-Larynx and Esophogeal Speech, J. Speech & Hearing Disorders 20:291-299 (Sept.) 1955.

Acetazolamide in Ménière's Disease

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The pathologic anatomy in Ménière's disease is largely unknown. In current hypotheses, allergic, bacterial, and metabolic disturbances are considered as etiologic factors. The picture as described by Hallpike and Cairns [1] is that of an endolymphatic idiopathic labyrinthine hydrops, or so-called glaucoma of the labyrinth (Jenkins, Portmann, and others). Furstenberg [2] supports the theory that the tissues involved in Ménière's disease have either an increased affinity for sodium or an unusual sensitivity to it. Graf and Poretti [3] conducted experiments on guinea pigs to determine the origin of the perilymph with the use of radioactive sodium as a tracer. They discovered that the perilymph does not originate from the endolymph, because there is a great difference in their respective sodium concentrations; the level in the endolymph was always several times lower than that in the perilymph. The existence of a fairly high sodium level in the scala vestibuli a short time after injection indicates that the perilymph does not originate from the cerebrospinal fluid; it has the same value as in the serum. The endolymph has quite a low sodium concentration. These findings favor the ultrafiltration theory; there must therefore be a barrier between the capillaries and the endolymph, and this can only be the epithelium of the stria vascularis. The observations of Waltner and Raymond [4] give additional support to the existence of a barrier membrane at the cochlear end of the acqueduct. The membrane does not permit diffusion of large colloid molecules but permits diffusion of crystalloids. Further investigations by Altmann and Waltner [5] proved that perilymphatic fluid is formed in the perilymphatic space, most likely from the blood vessels. The great permeability of the walls of the endolymphatic system makes it probable that a spread of electrolytes takes place from the endolymphatic fluid into the perilymphatic fluid, or vice versa. This would signify that both are virtually isoosmotic.

It can be, as Hilger [6] believes, that a vasomotor labyrinthine ischemia precedes the labyrinthine hydrops. There are spastic narrowing of the arteriole, reflux venous tonus, and transudation from the damaged capillary, resulting in compression of lymph capillaries by extravascular fluid. As there is no collateral circulation to remove the edema fluid, congestion is not relieved and normal circulation is not restored. Endolymphatic hypertension symptoms include vertigo, tinnitus, a sense of aural pressure, loudness, and diplacusis. Rarely, there is vomiting, nausea, and diplopia. According to Mann and Keilin,[7] carbonic anhydrase is present in every physiological barrier where ion exchange occurs, as in erythrocytes, gastric mucosa, pancreas, lungs, brain, kidney; Perkins [8] also found it in the iris and in ciliary tissue. Carbonic anhydrase is a zinc-containing enzyme (0.3%), obtained in a crystalline state (by Scott and Fisher [9]) and soluble in 99% alcohol. Its metabolic role is to preserve the alkalinity of sodium and potassium. According to Pitts [10] and Langham,[11] carbonic anhydrase acts to retain sodium in the aqueous humor, that which causes an increase in the intraocular pressure. Gloster [12] and Campbell [13] investigated the influence of sodium concentration in serum and changes in intraocular pressure. Perkins,[8] Gloster,[12] Langham,[11] Sourdille,[14] Palin,[15] Campbell,[13] and Duke-Elder [16] gave their views on glaucoma treatment with the

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use of a new carbonic anhydrase inhibitor: acetazolamide (Diamox) (Lederle). Among the numerous inhibitors of carbonic anhydrase Mann and Keilin [7] discovered in 1940 that the enzyme can be inhibited by small amounts of sulfanilamide (2×10^{-6} M). Schwartz [17] showed in 1949 that sulfanilamide would cause a diuresis in patients with cardiac failure. In the laboratories of the American Cyanamid Company was synthesized a new heterocyclic sulfonamide, acetazolamide, which inhibits carbonic anhydrase and appears to be a powerful diuretic. As was mentioned, it was very successfully applied in the treatment of glaucoma. Aware of the similarity of the development of glaucoma and that of endolymphatic hydrops of the labyrinth, we used acetazolamide in the treatment of Ménière's disease in 27 cases. The results are presented below.

Report of Cases

The patients were chosen from among the recalcitrant cases, with a clear picture of the disease and a well-elaborated differential diagnosis. At first labyrinthine reflexes are the criterion for determining the degree of improvement. I used three tests for this purpose: otolith function, cold microcaloric test, and the galvanic stimulation test. Vertigo, tinnitus, and deafness are mentioned as only subjective uncontrollable symptoms in the psychoneurotic patients, who are those with advanced Ménière's disease. The previous treatment almost always consisted of dietary restrictions and vasodilatation (procaine, nicotinic acid, epinephrine, etc.). The preparation acetazolamide was given by intravenous infusion and also perorally under the given posology. Additional treatment was not given in order to avoid interference from the action of other drugs. In the Table is presented a statistical representation of all the cases treated, and from them only four were chosen for detailed description.

Case 3.—A 43-year-old man, alcoholic, tobacco smoker, without an important anamnesis in regard to the present illness. Two years ago he had an epileptiform attack, which was repeated monthly with strong vertigo, nausea and vomiting, diminished hearing, and perpetual tinnitus. Treated by intravenous procaine and stellate ganglion infiltration. No improvement. Caloric test response diminished, galvanic response diminished, both air and bone conduction lowered, after nystagmus toward the left ear. After the patient was on a salt-free diet for three days we gave him 500 mg. of acetazolamide in 250 ml. of 10%

Results in Cases of Ménière's Disease Treated with Acetazolamide

Case No.	Duration of Illness	Microcaloric Test		Otolith Function		Audition	
		Before	After	Before	After	Before	After
1	6 mo.	—	++	—	=	=	=
2	1 yr.	—	+	—	=	—	—
3	2 yr.	—	+	—	+	—	—
4	1 yr.	=	+	—	+	—	—
5	8 mo.	—	++	—	+	—	+
6	1 yr.	—	+	—	+	—	+
7	2 mo.	—	++	—	++	=	+
8	3 yr.	—	=	—	=	—	—
9	3 mo.	—	+	—	+	—	+
10	5 mo.	—	—	—	+	—	—
11	1 yr.	—	+	—	+	—	—
12	1 yr.	—	+	—	+	—	—
13	3 yr.	—	+	—	+	—	—
14	1 yr.	—	+	—	+	—	—
15	4 mo.	—	++	—	++	—	—
16	6 mo.	=	+	—	+	—	=
17	1 yr.	—	++	—	+	—	+
18	1 yr.	—	+	—	+	—	=
19	9 mo.	—	+	—	+	—	—
20	6 mo.	—	+ — +	—	+	—	—
21	1 yr.	—	—	—	=	—	—
22	2 yr.	—	=	—	=	—	—
23	16 mo.	—	—	—	+	—	—
24	6 mo.	—	+	—	+	—	=
25	1 yr.	—	+	—	+	—	—
26	8 mo.	—	++	—	++	—	=
27	1 yr.	—	+	—	+	—	+

The meanings of the symbols are as follows: —, absence of reflex or improvement; =, very little response or improvement; +, response or improvement; ++, very good response or improvement.

dextrose. Infusion well tolerated, showing a quick relief of tinnitus, cessation of vomiting and vertigo, and a general feeling of well-being. He asked for food, and his appetite was copious, to his own astonishment. I gave him another infusion six hours later, and the medication was continued for the next week with one tablet every five hours. In spite of the order to remain in bed, the patient clandestinely walked about. Otolith reflex, caloric test, and galvanic response were soon normal, but deafness persisted. He was discharged for home treatment, which was continued for six weeks. No other improvement was observed.

CASE 12.—A 50-year-old man, nonalcoholic tobacco smoker, who had malaria for 30 years, developed pyorrhea and cryptic tonsillitis. Had loudness and vertigo, especially in the morning; tinnitus and diplacusis. Nystagmus; caloric test response was very weak. Galvanic test was refused because he had a phobia of electricity; air and bone conduction weak. The previous treatment (one year) unknown. An intravenous infusion of 500 mg. of acetazolamide every six hours, for a total of four injections, resulted in improvement. The peroral treatment was continued with a 750 mg. daily dose. In time tonsillectomy was done, and pyorrhea treatment continued. Two months later patient was discharged for home treatment. Five months later no signs of further attacks or tinnitus.

CASE 13.—A 37-year-old woman, nonalcoholic, nontobacco smoker, 3 partus, 2 abortus, got malaria three years ago, and treatment caused vertigo attacks. Her hearing was diminished, and sifflement accompanied her, even in "dreams." She was in a very miserable psychic state, and was narcoanalyzed without a special shell of psychosomatic origin in her affection. She had been previously treated with belladonna, scopolamine (Hyoscine), and barbiturates, with poor results. Suicide was attempted several times. Four infusions of acetazolamide improved her mental condition and brought about restful sleep, a good appetite, and cessation of vertigo and sifflement. The symptoms that had been present for three years disappeared all at once. The peroral therapy was interrupted twice because of drowsiness of the patient. She herself stopped the treatment on the sixth week, without recurrence of any of the mentioned symptoms. Caloric response was normal; otolith function present.

CASE 20.—Menopausal woman, 46 years old, 7 abortus, anamnesis not clear, Wassermann reaction negative, nonaddiction to drugs. Vertigo, nausea, tinnitus, temporary deafness, and a rare attack of vomiting in the morning brought the patient to my ward after six months of suffering. Previously treated by hormonal therapy (sterones). Galvanic test response very weak, caloric test weak, nystagmus and counternystagmus in rotating test. Daily infusion of acetazolamide and peroral therapy during four weeks relieved all subjective symptoms, but we continued treatment up till caloric and galvanic tests were normal, for four more weeks.

I cannot ignore the reaction of the patient in any treatment of Ménière's disease, for the therapeutic effect not only gives us evidence of the efficacy of the treatment but also a new look at the background of the physiopathological changes which occur in this disease. The role of the carbonic anhydrase in the function of the labyrinth is going to appear in a very different light. For the moment that is the problem I would point up. This finding, together with the previously mentioned historical reports on endolymphatic hydrops, and the behavior of carbonic anhydrase in other diseases of disturbed dialysis can elucidate many problems.

Comment

The possibility of direct response of carbonic anhydrase in the later development and pathological picture of endolymphatic hydrops is almost certain. The tests of qualitative and quantitative values of this enzyme in the membrane of the inner ear in normal and diseased ears, also the blood values of this enzyme in both cases, will certainly throw more light on the problem. Correlation between Furstenberg's [2] theory of the increased affinity of sodium in the tissue in cases of Ménière's disease and treatment of the disease with carbonic anhydrase inhibitors serves to substantiate my opinion that carbonic anhydrase is in the first stage responsible for sodium accumulation in the endolymph, causing osmotic traction of the endolymph toward the perilymph; condensation of the perilymph; increase in the volume of the endolymphatic fluid, and thus hypertension—all attributed to the changed physicochemical conditions in the striated endoderm, and disturbed osmosis. It is clear that these conditions also produce important pH concentration changes in the perilymph and endolymph, which can be also

very important in the development of further pathological changes in the surrounding tissues. The treatment of Ménière's disease with acetazolamide can be an important improvement in the therapy of the disease, accompanied by additional treatment in relation to vasomotor causes and anticoagulant drugs which prevent precipitation of fibrin in narrowed capillaries.

The hypothesis that a disequilibrium of autonomic innervation of the arterioles produces vascular spasm with resulting metabolic disturbances can explain the particular clinical picture of sudden deafness and vertigo in the young person apparently in good health. But if we apply this hypothesis to Ménière's disease (or idiopathic hydrops of the labyrinth) the existing evidence does not indicate that such an explanation is valid. Hoogland[18] believes that endolymphatic hydrops may be due mainly to disturbances in the vasomotor supply. According to Fowler,[19] vascular spasm and concomitant blood sludging may be caused by many factors, including emotional episodes. It is probable that persistent sludging may account for permanent deafness, tinnitus, and vertigo, which can be different from transient neural ischemic vertigo, tinnitus, and deafness.

The formation of perilymph originates in the capillary network of the stria vascularis, since no fluid current in the direction of the perilymphatic space could be demonstrated coming from either the subarachnoid or the endolymphatic space. This means that once the blood circulation is arrested in the perilymphatic space, by spasm or by thrombosis, the formation of perilymphatic fluid stops. Resorption of perilymphatic fluid takes place in three areas: from both scalae into the spiral ligament and from the vestibule and the semi circular canals into blood vessels in the trabecula of the perilymphatic tissue. The difference in the sodium content of perilymph and endolymph is great. The endolymphatic content in normal conditions is several times lower than that of perilymph, resulting in a positive osmotic pressure in the ductus cochlearis, which holds the physiological turgor of the endolymphatic sac. As the crystalloid molecules diffuse from the endolymph to the perilymph and vice versa, once formation of the perilymph is inhibited diffusion of salts through the endolymphatic sac stops after osmotic equilibrium on both sides of the membrane is established, and naturally starts a hideous hypertension in the endolymphatic sac, thus increasing its volume and decreasing the volume of the perilymph, resulting in an endolymphatic labyrinthine hydrops or Ménière's disease.

The therapeutic effect of acetazolamide in Ménière's disease consists in inhibition of carbonic anhydrase present in the endolymphatic sac, thus preventing retention of sodium in the endolymphatic fluid and at the same time lowering the osmotic pressure of the endolymph. It is also in accordance with the findings of Waltner and Raymond,[4] who found a rise in the sodium level of the endolymph in patients suffering from Ménière's disease. The results I found were such to encourage me to continue further laboratory and clinical investigation of this method.

Summary

Twenty-seven patients with Ménière's disease were treated with acetazolamide (Diamox) by intravenous infusion and by peroral therapy. In all cases the establishment of labyrinthine reflexes was observed during the treatment. Otolith function was also recovered; only the deafness problem, probably more complicated than the vestibular function, was not much improved. The mechanism of action is discussed.

The representative of Lederle Laboratories in Bagdad supplied the acetazolamide for our cases. The Director of the Middle Euphrates Hospital Groups, Dr. S. Baker Mussawy, helped in our experiments.

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REFERENCES

1. Hallpike, C. S., and Cairns, H.: Proc. Roy. Soc. Med. 31:1317, 1938.

2. Furstenberg, S.: Ann. Otol. Rhin. & Laryng. 43:1035, 1934.

3. Graf, W., and Poretti, G.: Pract. oto-rhino-laryng. 12:351, 1950.

4. Waltner, J. G., and Raymond, S.: Laryngoscope 60:912, 1950.

5. Altmann, F., and Waltner, J. G.: Ann. Otol. Rhin. & Laryng. 59:657, 1950.

6. Hilger, J. A.: Ann. Otol. Rhin. & Laryng. 59:1102, 1950.

7. Mann, T., and Keilin, D.: Nature 146:164, 1940.

8. Perkins, E. S.: Brit. M. J. 1:1273, 1955.

9. Scott, D. H., and Fisher, A. M.: J. Biol. Chem. 144:371, 1942.

10. Pitts, R. F.: A. M. A. Arch. Int. Med. 89: 864, 1952.

11. Langham, M.: Brit. M. J. 1:1273, 1955.

12. Gloster, J.: Brit. M. J.: 1:1273, 1955.

13. Campbell, D.: Brit. M. J. 1:1273, 1955.

14. Sourdille, G. P.: Brit. M. J. 1:1273, 1955.

15. Palin, A.: Brit. M. J. 1:1273, 1955.

16. Duke-Elder, S.: Brit. M. J. 1:1273, 1955.

17. Schwartz, T.: New England J. Med. 240: 173, 1949.

18. Hoogland, G. A.: Nederl. tijdschr. geneesk. 95:1798, 1951.

19. Fowler, E. P.: Ann. Otol. Rhin. & Laryng. 59:980, 1950.

Multiple Carcinomas in the Oral Cavity and Pharynx

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Malignancies originating in the oral cavity and pharynx comprise "about 4% of all human cancer and about 70% of all cancer in the upper respiratory and alimentary tracts."[1] Benign and malignant lesions involving the lips, buccal mucosa, tonsil, palate, and tongue are frequently seen by the otolaryngologist. Leucoplakia and patches of irritated mucosa, possible precursors of cancer, are examined daily. Many of these premalignant changes if observed over a period of time show evidence of neoplastic degeneration. Occasionally it can be demonstrated that areas of precancerous change progress to form multiple, separate areas of cancer.

Too often, despite the type and intensity of treatment, oral malignancies recur in the same location or in adjacent areas, or metastasize after the primary lesion is controlled. In many instances the recurrence is localized, without evidence of lymph node metastasis. In these cases the question arises whether the second cancer is recurrence of the primary or an entirely new malignancy. In each such instance it must be asked whether this represents local recurrence, submucosal spread of neoplastic tissue, mechanical transplantation of neoplastic cells, or multiple foci of malignant change. The possibility also exists that an entire area, through previous degenerative change in the mucosa, may have malignant propensities.

In the past separate different malignancies in one or more portions of the human body have been recorded. Maddock et al.[2] presented one case with four independent primary neoplasms—in the right breast, right ovary, sigmoid colon, and left breast. The individual tumors appeared over a period of time. A brief review of the literature pertaining to multiple neoplasms was also presented. In the esophagus Jackson[3] reported cases having apparent multiple centers of carcinoma. The possibility of submucosal spread of malignant cells was not ruled out.

Figi[4] reports that multiple malignancies in the oropharynx are rare. Drooker[5] stated that multiple cancer is found in 1.8% of all cancer cases. He observed a case having three separate, independent malignancies which occurred over a five-year period. These included a basal-cell carcinoma on the forehead, an epidermoid carcinoma in the larynx, and an adenocarcinoma in the nose. He also reviewed three other reports of triple carcinomata: One patient had three epidermoid carcinomas on the lips, another one epidermoid lesion in the larynx and two squamous-cell tumors in the esophagus, and a third patient had three separate epidermoid carcinomas on the tongue. Drooker also quoted Warren and Gates, who reviewed 115,000 postmortem examinations. Of these, there were 1259 with multiple malignancies. In 37 of the 1259, there were three separate malignancies (2.9%). Mumma[6] noted a case in which there were epidermoid malignancies, one on each anterior tonsillar pillar and one at the base of the tongue. Byers and Anderson[7] reviewed a series of 166 cases with oral cancer, all epidermoid carcinoma, varying only in the degree of differentiation. Of these, 20 (12%) had multiple lesions. Four cases had two or more cancers at the time of the original examination; sixteen developed second and subsequent lesions at some time after the appearance

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From the Radiology Department, Sioux Falls Clinic (Dr. Breit); Pathologist, McKennan Hospital (Dr. Myrabo).

of the original lesion. The time interval for appearance of subsequent carcinomas was from three weeks to seven years. Twelve patients had two carcinomas, five had three lesions, and three cases had four epitheliomas. Slaughter[8] showed that oral carcinomas have greater linear extent than extent in depth, the epithelium at the periphery of the neoplasm showing degenerative changes. He feels that his findings indicate a selective or guided-missile type of carcinogenic activity. By means of "field cancerization" individual malignancies arise from multifocal areas of precancerous change and not from one cell which suddenly becomes cancerous.

The fact that individual malignant tumors may have multiple potentialities has been discussed previously. Neoplasms with elements of squamous epithelium, columnar epithelium, and basal-cell types as well as apparent sarcomatous change, variously called adenoacanthoma, carcinosarcoma, collision-cell tumor, pleomorphic carcinoma, and adenocystic basal-cell carcinoma, have been described. In a previous paper one of us (J. B. G.)[9] reported a polypoid tumor in the midesophagus in which epidermoid, basal-cell, and adenocarcinoma and apparent fibrosarcoma elements were present. This tumor probably arose from tissue which had pleuripotential growth propensities.

Martin[1] reports that about 75% of oral cancer is the squamous-cell type and well differentiated (Grades 1 and 2). The less differentiated epidermoid carcinomas are found oftener in the posterior portion of the mouth (tonsil and base of tongue). In this location the tumors are frequently asypmtomatic, thus delaying diagnosis. These lesions often metastasize early, the metastatic node in many instances being the first evidence of abnormality. Pressman[10] observed that the entrance of neoplastic cells into the lymphatics or blood stream is influenced by pressure. In the mouth and throat the pressure produced by mastication and deglutition is a definite factor influencing early spread of a tumor. The first metastasis is usually to the neighboring lymph nodes of the same side. However, many neoplasms, especially those on the tongue, soft palate, and the posterior pharyngeal wall, may spread to both sides because of the bilateral lymph drainage. The neoplastic cells usually spread in the direction of lymph flow, but if there is obstruction to the lymphatics with disarrangement of flow, the malignant cells may follow an erratic pathway.

A knowledge of the complex lymphatic drainage from the various portions of the posterior oral cavity and pharynx is important in understanding treatment and the prognosis of malignant lesions in this area. There are multiple channels of egress for each individual area and much overlapping of the points supplied. The profuse supply of lymphatics, the tendency for bilateral spread of tumors, and the pressure produced by mastication and deglutition influence early spread of neoplasms from this area, making them much more treacherous than many other cancers.

The lymphatic drainage from the soft palate is rich, the vessels passing toward either surface of the palate. From the superior surface lymphatics join vessels from the nasal mucous membrane near the Eustachian tubal orifices and go to the superior deep cervical nodes. Other lymphatics penetrate the superior constrictor of the pharynx and pass posteriorly and superiorly into the retropharyngeal nodes. Lymphatics from the inferior surface of the soft palate descend beneath the mucous membrane covering the posterior pillars of the fauces, perforate the superior constrictor, and enter the upper nodes of the superior deep cervical group of nodes. Occasionally lymphatic stems from the soft palate terminate in the parotid[11,13] and subparotid nodes.[16]

The lymphatic egress from the tonsil is also abundant. The primary drainage is with stems from the basal portion of the tongue which pierce the pharyngeal wall and go to the superior deep cervical nodes, primarily to the jugulodigastric node near

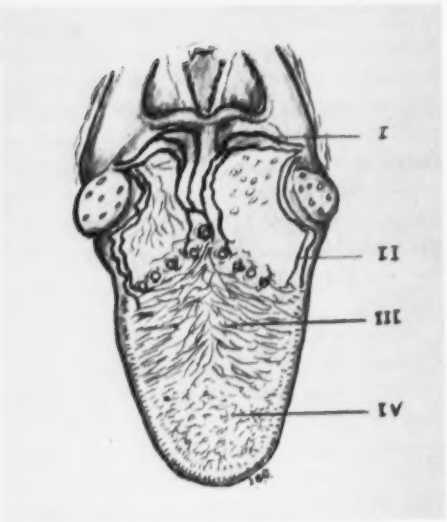


Fig. 1.—Submucosal lymphatics of the tongue: I, median basal lymphatics; II, lateral basal lymphatics; III, lateral network of lymphatics; IV, apical network of lymphatics. (Redrawn with permission from Piersol, Human Anatomy, Ed. 3, Philadelphia, J. B. Lippincott Company, p. 953.)

the angle of the jaw.[14,17,18] Other lymph vessels accompany the various tonsil arteries. Some branches accompany the lymphatics of the soft palate and go ultimately to the superior deep cervical lymph nodes.

Lymph drainage from the basal portion of the tongue is largely independent of that of the anterior portion (Fig. 1). It originates in the dense submucous network which surrounds the circumvallate papillae and the foramen cecum. There are usually four stems, two placed medially and two lateral stems. The medial vessels pass directly posteriorly and bend laterally in the glossoepiglottic folds to join the lateral stems near the tonsils. The lateral stems drain the region of the lateral circumvallate papillae, the foliate papillae, and the glandular region of the tongue. They are directed posteriorly toward the inferior border of the tonsil. After joining the medial branches, these lymphatic vessels perforate the lateral pharyngeal wall to enter the superior deep cervical nodes.[15] Lymphatics from the superior portion of the larynx, including the epiglottis, follow the superior laryngeal artery through the thyrohyoid membrane to the upper deep cervical glands, partially via the infrahyoid nodes in the prelaryngeal space.[12] However, the drainage from the anterior portion of the epiglottis may be directly to the upper deep cervical nodes along with lymphatics from the base of the tongue.

The submucosal bed of lymphatics is very rich throughout the oral cavity and pharynx, and there is much overlapping of the lymph supply of the various portions of the posterior part of the oral cavity. The complexity of the submucosal net of vessels undoubtedly is important in the local spread of malignant cells. In many instances it may account for the apparent multiple foci of cancer some distance apart. The "field cancerization" of Slaughter may be enhanced by the profuse submucosal lymphatic distribution of the pharynx.

Report of Case

A 70-year-old white man complained of the sensation of a lump in his throat when swallowing for two months. He had smoked about one-half package of cigarettes per day for many years.

Fig. 2.—Photograph of the soft palate showing two confluent neoplasms.

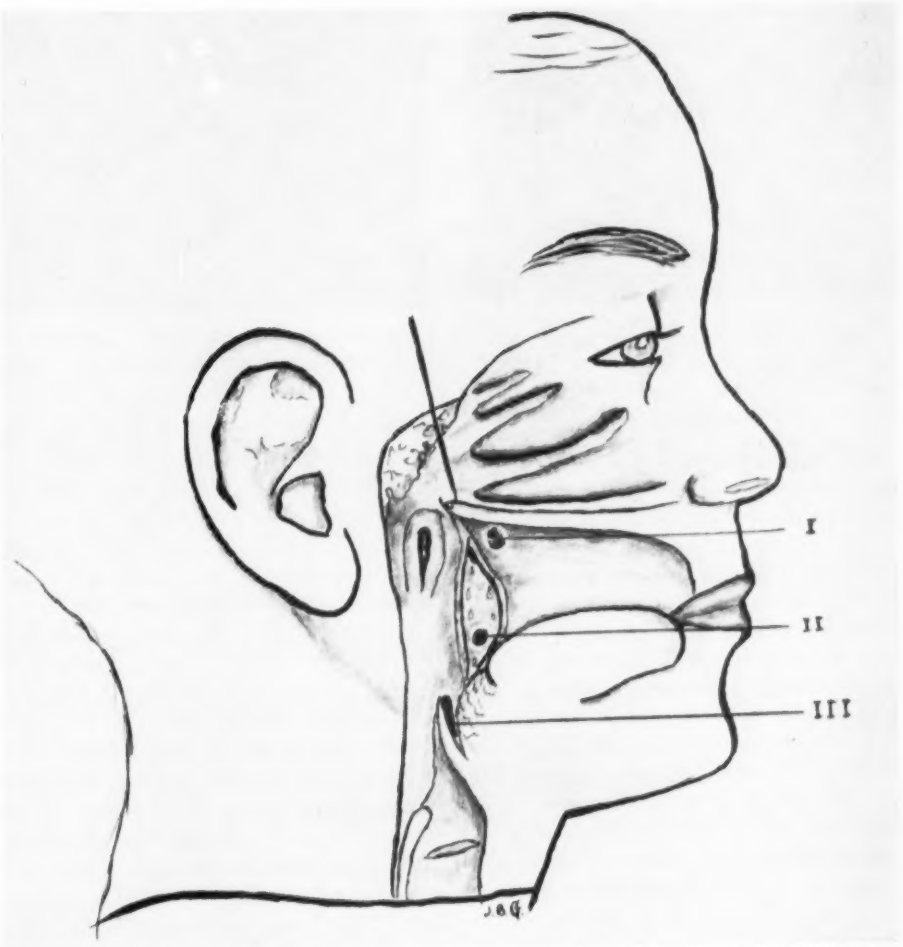


Fig. 3.—Lateral view of the oral cavity and pharynx showing the location of the individual tumors.

Past history and general physical examination were normal. Blood serology was negative. Examination of the mouth showed two well-demarcated, confluent, slightly raised papillary lesions (each 4 mm. in diameter) on the soft palate, slightly to the left of the midline (Fig. 2). On the surface of the left tonsil near the lower pole (Fig. 3) there was a circumscribed, raised papillary nodule 8 mm. in diameter. On the anterior surface of the epiglottis extending into the glossoepiglottic sulcus there was an irregular ulcer 1.5 cm. in diameter, the depths of which were covered with friable, granular-appearing, vascular tissue. The mucosa covering the remainder of the soft palate, posterior pharyngeal wall, and hypopharynx appeared atropic and chronically inflamed, suggesting pharyngitis sicca. No other lesions and no leucoplakia were present. Biopsy of the four lesions showed moderately well-differentiated squamous-cell carcinoma (Grade 2-3) in each specimen (Figs. 4, 5, and 6). The neoplasms were largely confined to the surface without much invasion of the underlying stroma. The epithelium at the periphery of the specimens showed hyperplasia and some acanthosis. In the surrounding stroma there was fibroblastic reaction and mild infiltration by lymphocytes. The patient received x-ray therapy to all lesions, with immediate good response of the four malignancies. The post-therapy follow-up period is short, so it has been impossible to determine whether he will have recurrence or spread.

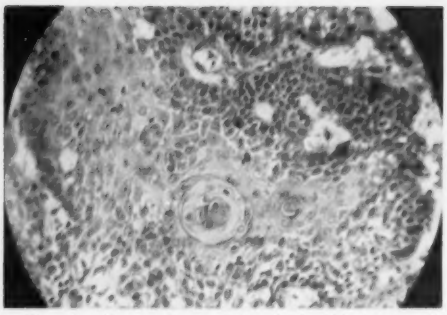


Fig. 4.—Photomicrograph of the biopsy specimen removed from the lesions on the soft palate, showing a moderately well-differentiated epidermoid carcinoma.

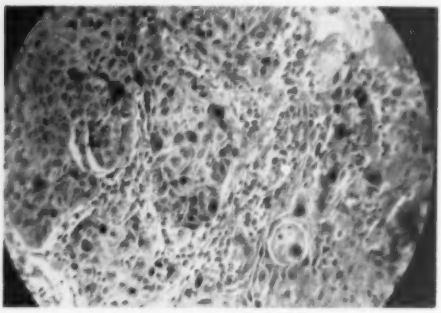
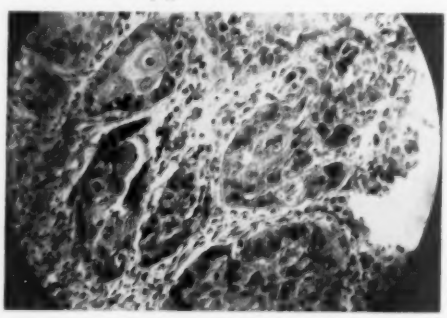


Fig. 5.—Biopsy specimen taken from the left tonsillar area. Note the histological similarity to Figures 4 and 6.

Comment

The possibility that one person may develop multiple malignant new growths has been known for a long time. Multiple new growths are found in about 2% of persons with cancer. The question arises as to what chemical or structural difference exists between this person and his fellow beings. Some factor which predisposes the formation of neoplastic tissue is present in this person. The difference could be one of "field cancerization," as described by Slaughter, in which an entire area of the body becomes disposed to malignant degeneration by means of chronic irritation or structural change in the mucosa due to age. It also seems that there must be an intrinsic factor, individual or genetic, influencing development and maturation of the constituent cells which influences the production of new growths. This latter concept receives support from cases such as the ones reported by Drooker and Maddock. Histologically different types of epithelial neoplasms were noted in the same person in different anatomical locations over a period of time. In other instances, such as the cases reported by Mumma, Jackson, Byers, Anderson, and Slaughter, squamous epithelial neoplasms which were histologically similar were present in different areas. Anatomical relation between the malignancies was unlikely. In the case presented here direct anatomical continuity between three of the four tumors was not demonstrable, although the lesions were on the same side of the throat. If the rich pattern of submucosal lymphatics and the direction of flow of these lymph channels in the throat are considered, it is possible that the apparently multiple tumors actually represented submucosal spread of malignant cells. The milking effect of mastication and deglutition could have been a factor in the local spread. However, the fact that the epithelium at the periphery of the individual biopsy specimens showed degenerative changes would suggest that the entire area had malignant propensities. This is in accordance with the findings in Slaughter's cases. Although submucosal lymphatic spread is a possibility, in this case it seems more likely that multiple individual cell units underwent malignant alteration in a fertile bed which had been liberally seeded by degenerative changes in the mucosa.

Fig. 6.—Biopsy specimen taken from the anterior surface of the epiglottis.



The important lesson to be learned from a case such as the one presented here is that there may be multiple areas of malignant change in the oral cavity of patients who have degenerative alteration in the mucosa. Many of the recurrences, seeming local spread and metastasis of neoplasms, following apparently adequate therapy can be accounted for on the basis of multiple individual areas of neoplastic change.

In the event a malignant tumor is found in the oral cavity prudence would dictate that any other area of alteration in the mucosa be evaluated carefully, preferably by biopsy, prior to institution of therapy. If degenerative changes are found in the mucosa, especially those suggesting carcinoma in situ, the field of treatment must be enlarged to include all suspicious areas.

Conclusions

1. Multiple individual malignant tumors may be found in various parts of the body. They may be of the same or different cell types.

2. In the oral cavity and pharynx multiple epithelial neoplasms are found simultaneously and at time intervals in locations not anatomically related.

3. Any multiple oral lesions which are histologically similar must be suspected of submucosal lymphatic spread, mechanical spread, local recurrence, or multiple individual foci of neoplasm formation.

4. In the case presented four individual squamous epithelial tumors were found. This apparently represents multiple foci of neoplasm in an area having malignant propensities by virtue of previous atrophic and degenerative changes.

Addendum

In March, 1957, this patient developed right lower lobe pneumonitis. At bronchoscopy there was severe generalized chronic bronchitis, and an intraluminal neoplasm was seen in the right lower lobe bronchus. Biospy showed a moderately well-differentiated epidermoid carcinoma, the fifth such lesion which this man has developed. Mediastinal involvement precluding resection was found at thoracotomy.

14 Riverview Hts.

REFERENCES

1. Martin, H.: Cancer of the Head and Neck, a Monograph for the Physician, American Cancer Society, Inc., Chicago, Lakeside Press, 1949, pp. 9-21.

2. Maddock, W. G.; Lipinski, J. J.; Lein, R. C., and Alt, H. L.: Multiple Primary Malignancies, A. M. A. Arch. Surg. 72:628-633 (April) 1956.

3. Jackson, C.: Carcinoma and Sarcoma of the Esophagus: A Plea for Early Diagnosis, Am. J. M. Sc. 169:625-648 (May) 1925.

4. Figi, F. A.: Diagnosis and Treatment of Malignant Tumors of the Throat, Mississippi Doctor 27:115-118 (Sept.) 1949.

5. Drooker, J. C.: Triple Primary Carcinoma in Otolaryngology, J. A. M. A. 109:1180-1183 (Oct. 9) 1937.

6. Mumma, C. S.: Multiple Lesions in Carcinoma of the Oral Cavity, Arch. Otolaryng. 52:104-107 (July) 1950.

7. Byers, L. T., and Anderson, R.: Multiple Cancers of the Oral Cavity, Am. Surgeon 18:386-391 (April) 1952.

8. Slaughter, D. P.; Southwick, H. W., and Smejkal, W.: "Field Cancerization" in Oral Stratified Squamous Epithelium, Cancer 6:963-968 (Sept.) 1953.

9. Gregg, J. B., and Stamler, F. W.: Unusual Neoplasms of the Esophagus: Review of Literature and Report of Case: A. M. A. Arch. Otolaryng. 59:159-169 (Feb.) 1954.

10. Pressman, J. J.: Extended Retrohyoid Radical Neck Dissection for Cancer of the Oral Cavity and Neck, Surg. Gynec. & Obst. 100:329-344 (March) 1955.

11. Grant, J. C.: A Method of Anatomy, Ed. 2, Baltimore, Williams & Wilkins Company, 1940, p. 724.

12. Reference 11, p. 725.

13. Piersol, G. A.: Human Anatomy, Ed. 3, Philadelphia, J. B. Lippincott Company, 1911, p. 954.

14. Reference 13, p. 955.

15. Reference 13, p. 953.

16. Gray, H.: Anatomy of the Human Body, revised and reedited by W. H. Lewis, Ed. 23, Philadelphia, Lea & Febiger, 1936, p. 690.

17. Reference 16, pp. 690-691.

18. Thorek, P.: Anatomy in Surgery, Philadelphia, J. B. Lippincott Company, 1951, pp. 164-165.

Indications and Predictions in Stapes Mobilization

New Method to Test Hearing During Surgery

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In the otosclerotic type of deafness the footplate of the stapes is fixed in the oval window, thereby impeding transmission of sound to the inner ear. Following unsuccessful attempts toward the end of the last century, a new surgical procedure to free the ankylosed base of the stapes and reestablish the physiologic route of sound transmission was proposed by Rosen.[10,11] The operation has been accepted by some surgeons and rejected by others, and the controversy is bound to continue until the long-term effectiveness of the procedure can be evaluated. In the meantime various attempts have been made to improve the surgical technique, to clarify the indications for stapes mobilization, and to arrive at more precise predictions.[3,6,8,13,15]

In theory it might be expected that with restoration of the transformer action of the middle ear hearing will return to the bone-conduction level. However, the hearing is often postoperatively stabilized below the bone-conduction level, in spite of successful mobilization of the stapes. This may be due to any one of a number of causes: diminished tension of the drum membrane, scar formation, organized clots in the drum or middle ear which interfere with conductive properties and resonance, an irregular fracture line of the footplate, or escape of perilymph. Nevertheless, selection of cases and evaluation of results must be based on the preoperative level of bone conduction.

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Indications

Best results are obtained in the group of cases where the loss of bone conduction is not greater than 15 db.; this is the category in which fenestration operation is also highly successful. However, there is an important difference between the indications for the two procedures: Fenestration operation is most effective when the gap between the air- and bone-conduction curves amounts to at least 30 db. in some of the speech frequencies; stapes mobilization, on the other hand, is feasible with an air-bone gap of not more than 20 db. Thus stapes mobilization would seem to be indicated during an earlier stage of otosclerosis, without precluding subsequent fenestration in case of surgical failure, or in later recurrence of the disease.

A second group of cases in which stapes mobilization is the procedure of choice are those where bone conduction is between 15 and 30 db.; in this group complete ankylosis of the stapes is the rule and there is a wide air-bone gap.

In a third group of patients the loss of bone conduction is so great that restoration of serviceable hearing can no longer be expected. However, stapes mobilization is still useful, as it is apt to bring the very depressed air-conduction curve up to a level where the patient may be able to use a hearing aid to good advantage. In these cases of advanced stapes ankylosis combined with nerve degeneration, it is often possible to eliminate the conductive element of the hearing impairment by mobilization.[14]

Stapes mobilization may be considered in still another category, i. e., where fenestration operation has failed to produce serviceable hearing. Previous fenestration does not rule out subsequent stapes mobilization. But since the incus has been removed it is necessary to create columella action by retropositioning a large tympanomeatal flap. Unfortunately, after fenestration operation it is often impossible to fashion a sufficiently large tympanomeatal flap, and tympanoplasty should therefore be performed with a free skin graft covering the stapes.

Finally, stapes mobilization is indicated to reestablish bilaterally equal hearing. It has been shown that the separation of speech from background noises depends on the ability to locate the source of the competing sounds, which in turn can be accomplished only when reception is about equally good on both sides.[7]

In reviewing the indications for stapes mobilization it becomes clear that this operation is more widely applicable than fenestration; it can be performed at an earlier stage of otosclerosis as well as in advanced cases when fenestration is no longer feasible. Differences between the various groups in which stapes mobilization is indicated are so pronounced that it is impossible to use the same basis for an estimate and prediction of the probable outcome of the intervention. In general the prognosis depends upon the preoperative level of bone conduction, the expectation being that following successful mobilization of the stapes air-conduction values should come up to bone-conduction values. However, this applies only when the preoperative bone-conduction loss does not exceed 30 db., only then is there a good chance that hearing will return to a serviceable level. In profound deafness such an expectation is unwarranted and the operation has a more restricted aim. We are inclined to quote somewhat more favorable statistical figures to younger patients because for them the expectation of success is greater than for those in the older age groups.

Results

In view of the great importance of the preoperative bone-conduction level for selection as well as prediction, 100 cases have been analyzed by dividing them into the following three categories:

Group 1: Bone conduction between 0 and 15 db.
Group 2: Bone conduction between 15 and 30 db.
Group 3: Bone conduction above 30 db.

But as the therapeutic aim is not quite the same in the different categories, a uniform interpretation of the postoperative results would be misleading. In the first two categories the operation is considered a success only if hearing is raised close to the bone-conduction level or at least serviceable hearing has been restored. In the category with a preoperative bone-conduction loss of 30 db. or more, no such improvement can be expected and cases are classified as successful even when hearing does not return to the serviceable level, provided the patient is able to obtain greater benefit from a hearing aid.

Among 27 patients with a preoperative bone-conduction loss of 0-15 db., hearing improved in 51.85% of cases; this figure is considerably lower than the results obtained by means of fenestration in the same category. Among 52 patients with a loss of 15-30 db. the rate of improvement was 36.54%. The over-all improvement in these two categories amounts to 41.77%.

Among 21 patients with a preoperative bone-conduction loss in excess of 30 db. stapes mobilization was considered successful in 28.57%, using the less rigid standards commensurate with this advanced degree of deafness; hearing returned to the serviceable level in only one case, in which bone conduction improved too.

Technique

It is commonly agreed that success of stapes mobilization depends on an impeccable surgical technique.[16] In view of the many anatomic variations encountered in the middle ear, the procedure should always be planned so as to allow for unforeseen

emergencies. The first prerequisite is satisfactory exposure. It may be necessary to remove some of the overhanging posterosuperior wall of the canal to bring the incudostapedial joint and part of the footplate into view. Under the dissecting microscope, shape, arrangement, and angulation of the incudostapedial junction are carefully examined and the light pastel blue color of the footplate is noted. Any variations of the incudostapedial joint must be taken into account before deciding upon the proper direction of force required to loosen the footplate in the oval window.

During the operation it can be observed that the movements of the stapes are not uniformly the same in all cases.[17] Ordinarily the footplate moves like a piston, but sometimes it is more like a rocking around the short or the long axis. Even after the footplate has been mobilized, the stapes may move only in a single direction, but as a rule free movement in all directions can be established.

At present it cannot be predicted how much force is required to mobilize the stapes. Sometimes very slight pressure will loosen the ankylosed plate, but occasionally even the lightest touch is sufficient to break the neck or crura. In the adult,[9] the stapes has gone through "profound skeletal emaciation, and to make matters worse, the ossicle is weakest where the otologic surgeon would like to find it strong, that is, at the basal extremity of each crus."[1] A sound like the snapping of a fingernail is a very ominous sign and usually means an unintended fracture. If the degree of pressure which can be safely exerted on head or neck is insufficient to mobilize the stapes, the force is directly applied to the footplate. This used to be a measure of last resort, but recent experience has shown that the approach is sound,[12] and some surgeons are even starting the maneuver at the footplate.[4]

In performing stapes mobilization the surgeon is guided by repeated audiometric readings.[5] For these hearing tests we use a fine sound probe (Zöllner) built into a bone conductor (Fig.). Any audiometer and its bone conductor will serve the purpose. The sound probe was first employed in tympanoplasty and proved so satisfactory that it is now also routinely used in stapes mobilization. Contact is made with any visible part of the stapes or the lenticular process of the incus, and readings are taken in the 500, 1000, and 2000 frequencies, before and after mobilization. Patients are instructed to disregard vibrations and to signal only the actual hearing of pure tones. This test is neither an air-conduction nor a real bone-conduction study, and might be called a "contact audiogram." Each reading takes only a few seconds and furnishes immediate comparative information as to the degree of mobilization achieved.

Bone conductor with built-in sound probe for "contact" audiograms during stapes mobilization.

In the course of mobilization of the stapes even the most seasoned endaural surgeon may run into trouble. If during manipulation the incudostapedial joint is disrupted, the lenticular process of the incus is gently placed back on the head of the stapes, and spontaneous healing is the rule. In order to prevent any pull on the stapes while restoring the incus to its normal position, the stapedial tendon is severed. In two of the cases of disrupted incudostapedial joint it was noted that the lenticular process was pulled away from the head of the stapes by traction of the posterior ligament and the incudomalleolar joint. With proper repair, hearing often improves to the bone-conduction level; failure to repair the incudostapedial joint will cause a loss of 25-30 db. or more. Thus, when it is impossible to adjust the incudostapedial joint, the incus is removed and the tympanomeatal flap retropositioned to the head of the stapes, in order to create a columella action. This is also indicated when part or all of the incus is missing—not a rare finding in middle-ear exploration.

In attempting to loosen the footplate, particular caution is required when using a piston-like movement. The stapes can easily be pushed into the vestibulum, an accident signaled by violent dizziness and nausea. Even if the stapes is immediately pulled back the result might be a considerable hearing loss by air as well as by bone conduction, but fortunately this complication is rare. A milder degree of hearing deterioration may follow fracture of neck or crura of the stapes, which is especially regrettable when the ankylosis was not yet complete. Sometimes a fracture will not interfere with the success of the operation, if the footplate is also cracked; this can probably be explained on the basis of the anatomical and physiologic findings of Basek and Fowler.[2]

Bilateral stapes mobilization in one session, as now routinely performed by me, increases the chances of success. To this practice there is only one exception: if damage to the labyrinth is noted while operation on the first side is in progress. Among the last 20 cases there were 18 bilateral stapes mobilizations; in one instance the procedure had to be interrupted on account of violent dizziness, and another patient had previously requested to be operated on on one side only. Following bilateral operation all patients were highly satisfied. Most of them recovered good hearing at least in one ear and did not mind very much if hearing remained poor in the other. As long as the procedure was limited to one side at a time, any failure was likely to be quite disappointing and discouraging to patient and surgeon alike. Some patients even developed a real antagonism against the surgeon and were difficult to convince that a second attempt at stapes mobilization, or fenestration operation, might bring better results. There are other reasons in favor of the bilateral operation, such as economic factors, only one hospitalization and one sick leave.

Comment

Stapes mobilization can be correctly evaluated only as one step in an integrated program of surgical rehabilitation of conductive hearing loss. It is not intended to take the place of fenestration operation, and its usefulness is enhanced by adapting certain features of other procedures, particularly tympanoplasty. With this flexible technique the endaural surgeon is better able to adjust to unforeseen anatomic variations and unexpected complications.

In typical, not-too-far advanced cases of otosclerosis the hearing impairment is due to partial fixation of the stapedial footplate. These are mainly younger patients with symptoms of relatively short duration, in whom an attempt to reestablish physiologic sound transmission is indicated. The results of surgical mobilization in this category are more satisfactory than in older patients, particularly with the technique of bilateral correction in one session. Return of hearing to the bone-conduction level is frequent, and when the procedure is performed during an inactive stage of the disease, what seems to be lasting improvement is obtained. If a first attempt should fail, the mobilization

operation can be repeated or, if preferable, fenestration may be performed. Stapes mobilization has the added advantage that tinnitus disappears in 50% of cases.

Stapes mobilization, however, is also beneficial in the more advanced phases of otosclerotic disease. It may restore binaurally equal hearing, eliminate the conductive component in far-advanced otosclerosis, and in profound deafness bring air conduction back to a level where the patient is better able to tolerate a hearing aid.

Surgical mobilization of the ankylosed stapes is thus useful in different phases of otosclerotic disease, and success of the procedure depends largely on correct selection of cases. Greater familiarity with this relatively simple operation is likely to extend its application, thus making the benefits available to an ever larger number of otosclerotic patients.

The fact that stapes mobilization is a short intervention entailing little hospitalization and a minimum of morbidity should not obscure the need for precise surgical technique. It stands to reason that not all details of this fairly new operation have been worked out, and further refinements of the precedure are desirable and will lead to increasingly better results.

Summary

Stapes mobilization is of great value in various phases of a surgical rehabilitation program for the otosclerotic patient. Success depends on correct selection of cases; the indications and predictions for surgery are discussed in detail.

The technique has been improved by introduction of "contact" hearing tests during mobilization, and by use of a skin graft to produce a columella effect when physiologic sound transmission cannot otherwise be recovered. Bilateral operation in one session has increased the number of successful cases. In carefully selected patients stapes mobilization will result in normal auditory acuity, or at least in a return of hearing to the serviceable level in around 40-50% of cases.

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REFERENCES

1. Anson, B. J., and Bast, T. H.: Development and Adult Anatomy of the Auditory Ossicles in Relation to the Operation for Mobilization of the Stapes in Otosclerotic Deafness, Laryngoscope 66:785-795 (July) 1956.

2. Basek, M., and Fowler, E. P., Jr.: Anatomical Factors in Stapes-Mobilization Operations, A. M. A. Arch. Otolaryng. 63:589-597 (June) 1956.

3. Bellucci, R. J.: Present Status of the Operation for Mobilization of Stapes, Laryngoscope 66:269-292 (March) 1956.

4. Fowler, E. P., Jr., in Symposium.[16]

5. Goodhill, V.: Trans-Incudal Stapedolysis for Stapes Mobilization in Otosclerotic Deafness (Under Audiometric Control), Laryngoscope 65: 693-710 (Aug.) 1955.

6. Kos, C. M.: Transtympanic Mobilization of Stapes for Impaired Hearing Due to Otosclerosis, Ann. Otol. Rhin. & Laryng. 64:995-1008 (Dec.) 1955.

7. Koenig, W., cited by Rosen and Bergman.[13]

8. Meurman, Y., and Meurman, O.: Stapes Mobilization in Otosclerosis: Primary Results and Review of 63 Cases, A. M. A. Arch. Otolaryng. 62:164-172 (Aug.) 1955.

9. Richany, S. F.; Bast, T. H., and Anson, B. J.: The Development of Adult Structure of the Malleus, Incus, and Stapes, Ann. Otol. Rhin. & Laryng. 63:394-434 (June) 1954.

10. Rosen, S.: Mobilization of the Stapes to Restore Hearing in Otosclerosis, New York J. Med. 53:2650-2653 (Nov. 15) 1953.

11. Rosen, S.: Simple Method for Restoring Hearing in Otosclerosis: Mobilization of Stapes; Case Reports, Acta oto-laryng. 44:78-88, 1954.

12. Rosen, S., in Symposium.[16]

13. Rosen, S., and Bergman, M.: Restoration of Hearing in Otosclerosis by Mobilization of the Fixed Stapedial Footplate: An Analysis of Results, Laryngoscope 65:224-269 (April) 1955.

14. Rosen, S., and Bergman, M.: Functional Goals in Mobilization of the Stapes, A. M. A. Arch. Otolaryng. 63:11-20 (Jan.) 1956.

15. Scheer, A. A.: Restoration of Hearing in Otosclerosis by Transtympanic Mobilization of the Stapes, A. M. A. Arch. Otolaryng. 61:513-534 (May) 1955.

16. Symposium: The Operation for the Mobilization of the Stapes in Otosclerotic Deafness, Laryngoscope 66:729-784 (July) 1956.

17. Wever, E. G., and Lawrence, M.: Physiological Acoustics, Princeton, N. J., Princeton University Press, 1954, pp. 111 and 155.

18. Thullen, A.: Die Schallsonde: Experimentelle und klinische Erhahrungen mit der Schallsonde nach Zöllner, Stuttgart, George Thieme Verlag, 1955.

Self-Recording Threshold Audiometry and Recruitment

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The measurement of auditory thresholds is ordinarily made by presenting the subject with tones of sufficiently varying intensity to obtain a point where he reports hearing the tone in 50% of the presentations. Although the number of given stimuli is generally not large in routine clinical work, the procedure, nevertheless, is a statistical measurement: The threshold is expressed as the mean of the observations between the intensities of the just-heard tone and the just-not-heard tone. The threshold is not simply the single figure expressed in decibels but an area around this figure with a width of 5 to 15 db., depending mostly on the mode of the stimulation [1] and on the reaction time of the subject.

The area between 0 and 100% responses to the stimuli can be charted with all methods of tone presentation. However, the method of limits and the method of constant stimuli are quite time-consuming for this purpose. The self-recording adjustment method described by Békésy [2] in 1947 affords a quick and simple means of revealing the width of the threshold area at various frequencies.

Interest in self-recording audiometry has increased considerably during the last years, although most of the work is still done for experimental purposes. The clinical interest has arisen in connection with diagnosis, since small excursions are reported to be associated with recruiting perceptive deafness.[2-4] However, most of the clinical reports have been marred by the fact that the presence of recruitment, and small threshold excursions (as a measure of the intensity difference limen) have been assumed to be paralleling phenomena, and a necessary correlation with direct recruitment testing has been omitted.

It was pointed out earlier [5,6] that the Békésy audiogram is rather a measure of the variability around the threshold and, as such, is mainly influenced by the growth of loudness. This is strongly supported by the fact that the width of the threshold excursions, about 10 db., closely corresponds with the intensity area for 0 and 100% responses with any other method of threshold determination. The size of the difference limen near threshold, on the other hand, should be of the order of 3 to 4 db.[7]

Review of Earlier Studies

Threshold Excursions in Normal Subjects.—An increase in the rate of change in intensity in a self-recording audiometer results in somewhat increased threshold excursions. Békésy [2] reported a change from 90 db. per minute to 220 db. per minute increased the width of the threshold from about 10 to about 15 db. At a rate of 140 db. per minute in 2 db. steps, the normal threshold variability was between 10-15 db. and the time needed for testing one ear 15 minutes.

Lundborg [3] also used a rate of 140 db. per minute in 2 db. steps and observed that in the majority the normal threshold varied between 6 and 9 db. The extreme lower limit was 5 db. and the upper 20 db.

In a paper by Reger and Kos [8] on a temporary threshold shift during continuous stimulation, results obtained on one normal subject tested for an hour's period were reported. At 250 and 1000 cps the thresh-

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old tracings showed a width from 4 to 6 db., while at 4000 cps the variability was only about 2 db. In this case the rate of change in intensity was 60 db. per minute with continuously varying attenuation. In a later paper Lierle and Reger[9] reported that attenuation of 120 db. per minute was used with the same apparatus and the few normal cases presented showed excursions of 4 to 10 db.

Although Békésy,[2] in his self-recording audiometry, employed both a fixed and a continuously changing frequency technique, it was the latter that he used in his clinical cases. A varying frequency technique was also used by Lundborg,[3] while Reger and Kos,[8] and Lierle and Reger[9] employed the old experimental technique of recording the threshold excursions at a fixed frequency as a function of time. Obviously the latter gives more precise data, as there is only one variable, intensity, instead of two, intensity and frequency.

My own experience with a self-recording audiometer[6] using the rate of change of 140 db. per minute in 1 db. steps, and a fixed frequency technique, indicated that the lower limit of the tracings only rarely is less than 5 db. Inexperienced subjects may easily show tracings of nearly 20 db. width, but in subsequent trials lower values, around 10 db., were generally obtained.

Threshold Excursions in Deaf Subjects. Békésy[2] did not state any definite limits of threshold variability for cases to be classified as recruiting. Rather, he was of the opinion that diminished excursions at the higher frequencies, as compared with the larger swings in the lower range, mean recruitment. In the two pathological cases reported the tracings were about 3 to 5 db., while in an experimental reversible lesion the tracings remained between 4 and 8 db.

Lundborg[3] went into much more detail in specifying which case was recruiting and which was not. The first category was that of normal excursions, viz., 6 to 9 db., and a tracing of this amplitude implied that there was no recruitment. The second group showed "a tendency to recruitment" in the form of somewhat diminished excursions at the higher frequencies, while in the third, that of "moderate recruitment," the excursions in the higher range decreased from 3 to 5 db. Only the fourth group showed "complete recruitment," and the width of the excursions was not more than 2 to 3 db.

Lundborg's clinical material supported his criteria fairly well. All 25 cases of acoustic trauma and practically all of the 26 cases of Ménière's disease belonged to the fourth group. On the other hand, 18 of 21 cases with retrocochlear pathology showed excursions characteristic of the first group with no recruitment. However, some of the results remained obscure: Owing to the lack of masking in air-conduction tests some curves were clearly shadow audiograms from the better ear. Also, the failure to use direct recruitment tests leaves open the question of recruitment function in each case.

Reger and Kos,[8] too, took it for granted that small excursions mean recruitment. This was demonstrated in two cases with small tracings. However, a case with verified acoustic tumor was also reported where the excursions at 1000, 2000, and 4000 cps were clearly less than 5 db. and, in fact, comparable with a recruiting case presented earlier (Reger and Kos,[8] Fig. 4, lower).

In a recent paper[6] I described the results obtained by comparison of the results of direct recruitment testing and the width of the automatic threshold recordings. It was concluded that, while a small threshold variability is generally accompanied by recruitment, not all cases with recruitment show small excursions in the threshold audiogram. This was the case, especially if the recruitment function was delayed and also in many cases with straight-line recruitment.

When threshold audiometry is done by recording the variability of the threshold as a function of time at a fixed frequency, the variability does not change much even if longer periods are used for continuous

stimulation.[8] However, a change of the order of ±5 db. should still be considered normal.[2,6] In clinical material this perstimulatory adaptation (or fatigue) at threshold can sometimes be much larger, even up to 30 db.[6,8,10] For a more comprehensive review of these phenomena, however, the reader is referred to an earlier publication.[6]

When continuous threshold recording is used as a guide to an evaluation of the loudness function in pathological cases, it is important to know the normal variability under various experimental conditions. Using the variable frequency technique, Lundborg[3] specified the size of the excursions suggesting the presence of recruitment. With a fixed frequency presentation as a function of time, only few normal results have been reported. Therefore it seems desirable to report on the normal variations observed during the last year, and also to discuss in this connection the relation between small threshold variability and recruitment function.

Experimental Procedure

The rate of change in intensity was the same as that used by Békésy[2] and Lundborg,[3] viz., 140 db. per minute in 1 db. steps. The patient operated the audiometer in the usual manner, by pressing and releasing a push-button which regulated the instantly reversible motor connected with the recording apparatus in an adjacent control room. The fixed frequencies were obtained from a Peters SPD 2 audiometer connected to the tone channel of the self-recording audiometer. The threshold recording on normal subjects was done for a one-half minute period at each frequency using continuous tones, and sharply interrupted tones at the rate of 1, 2, 4, 8, and 12 times per second with a constant on-off ratio of 0.5. The intensity of the tones before the attenuators was controlled with a Pegel voltmeter.

It was decided to use one highly experienced observer for repeated audiograms to see whether the variations in threshold amplitude changed noticeably in successive testing sessions. During two months' time 10 full sessions were recorded.

For the main group of observers 16 ears were used. Some of the subjects were quite familiar with the self-recording technique; for others this was the first testing. It was thought that this difference in experience might cause sufficient variation in the records and reveal the smallest and largest widths of excursions obtainable in normal subjects.

In the main group, testing was always begun with continuous tones, proceeding systematically from the repetition rate of 1 time per second up to the rate of 12 times per second. The highly experienced subject was presented with tone series at random.

In the pathological cases air- and bone-conduction audiometry was done in the usual manner. Recruitment testing was done by employing one of the direct tests, Fowler's or Reger's, as the case permitted. At the frequencies tested for recruitment, a fixed-frequency self-recording chart of the threshold was made during four minutes' continuous stimulation. In all tests masking with white noise was employed,[11] when necessary.

Results

Subjects with Normal Hearing.—The results of the threshold recordings on the highly experienced person and on the group of observers are presented in the Table. The Table shows the mean values of threshold variability, from peak to peak, and the standard deviations. It is seen that in both groups the mean variability is practically the same and that the width of the amplitude differs insignificantly at various frequencies. It also appears that the use of interrupted tones offers no advantages over continuous tones as far as the amplitude of the tracings is concerned.

Further, it may be noted that the standard deviations in the experienced subject are quite small and indicate little variation in

Mean Amplitude of Threshold Excursions and Their Variability

Subjects	Repet. Rate	125		250		500		1000		2000		4000	
		M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
One person (10 measurements per frequency)	Cont.	7.7	1.8	8.7	1.6	9.1	1.5	8.4	1.1	7.8	1.1	7.3	0.7
	1/sec.	9.0	2.0	9.5	1.8	9.6	1.9	8.6	0.8	8.9	1.2	8.7	0.7
	2/sec.	8.6	1.6	9.5	1.3	9.6	1.1	8.6	1.6	8.9	1.3	8.4	1.1
	4/sec.	9.3	1.4	9.4	1.3	9.5	1.3	8.2	1.2	8.0	0.8	7.6	0.7
	8/sec.	8.5	1.6	9.2	1.4	9.5	1.4	8.5	1.2	7.9	0.7	7.4	1.0
	12/sec.	8.9	2.1	9.1	1.8	9.4	1.1	8.7	0.9	7.7	0.6	7.3	0.9
Other subjects (16 ears)	Cont.	8.7	4.1	9.4	4.3	9.7	3.9	8.5	4.0	9.2	4.2	8.4	3.5
	1/sec.	10.1	4.3	10.3	4.6	10.4	4.7	10.1	4.6	9.7	4.1	9.7	4.9
	2/sec.	9.7	4.0	9.8	3.6	9.5	3.4	9.4	4.0	9.6	3.8	9.4	4.2
	4/sec.	10.2	4.4	9.5	4.0	9.9	4.1	9.6	4.0	9.5	3.8	9.1	4.1
	8/sec.	10.2	3.8	9.6	4.4	10.4	4.3	9.9	4.6	10.0	4.7	9.1	4.3
	12/sec.	10.1	3.9	9.7	3.8	10.0	4.3	8.4	3.9	8.8	4.2	8.2	4.1

the width of the tracings in the subsequent test. On the other hand, in the group of observers the standard deviations are clearly larger, as may be expected on the basis of the heterogeneous composition of the group. Especially the larger tracings, deviating considerably from the mean, tended to make the SD's bigger (Fig. 2) in this group.

It may be said that on the average the amplitude of the tracing varies from 7 to 10 db. regardless of whether continuous or 1- to 12-per-second interrupted tones were used as stimuli. The mean values, together with the mode and median, and the range of observations are also recorded graphically in Figures 1 and 2.

Figure 1 shows that the mean, mode, and median are nearly equivalent at all frequencies. The range of observations is quite small, especially at 4000 cps, and none of the values are below the 5 db. line. For the group of observers in Figure 2, both the mode and the median are consistently smaller than the mean, indicating that the

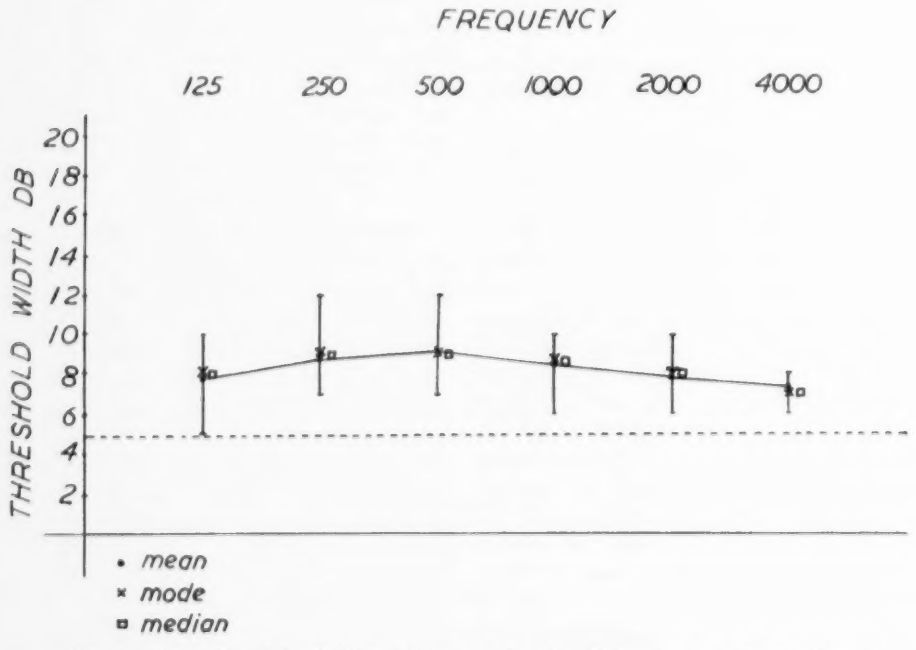

Fig. 1.—The width of threshold variability as a function of frequency on one experienced test person. The mean, mode, and median of 10 observations are all close together. The vertical lines indicate the small range of observations.

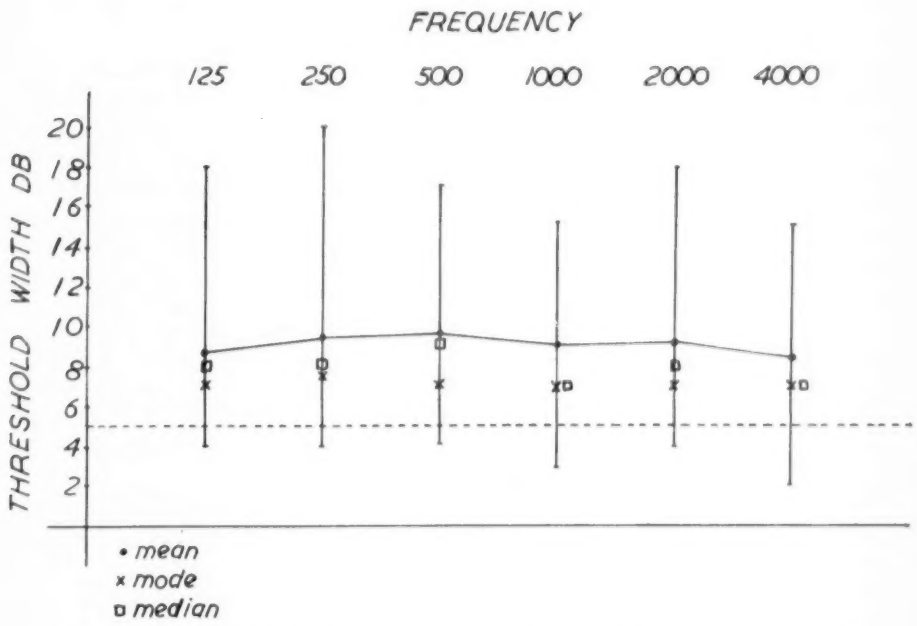

Fig. 2.—The width of threshold variability as a function of frequency in 16 ears. The mode and median are clearly smaller than the mean. The range of threshold excursions is large.

majority of the thresholds were below the mean value. The range of observations is quite large, from 2 to 20 db.

Very small excursions were found in only one test person, who showed them characteristically on all test occasions. In later control studies this person always showed tracings between 2 to 3 db. between 1000 and 4000 cps, while at 500 and 250 cps the values were somewhat larger, from 4 to 6 db. Apart from this one exception, the lowest values found in the group of observers were 5 to 6 db. Most of the inexperienced observers showed larger tracings, 15 to 20 db., and the experienced subjects more frequently showed tracings clearly below 10 db. It was also noted that in some cases the tracings diminished with experience, while in some, even with large amplitudes, training apparently had no effect.

On the basis of the SD values for the experienced observer, one would estimate that in about 1 out of 20 normal subjects the result will be below the 5 db. limit. This seems to agree quite well with the clinical testing experience.

Apart from these, some cases have been tested at the laboratory in which, in spite of efforts at training, only large tracings, of the order of 20-30 db., were obtained. These values are not necessarily related to the social status or level of education, and, as pointed out in a discussion by Reger,[8] it is sometimes surprising to see how well many rather simple people manage the self-recording technique. However, it is clear that for people with little technical experience the excursions on an average tend to be larger than normal.

It may be concluded that smaller than 5 db. threshold excursions are quite rare in normal persons. In general, therefore, smaller than 5 db. excursions can be used in the differential diagnosis, provided that the various clinical subgroups show significant variation.

The effect of various repetition rates upon the absolute thresholds of the tones as compared with those for continuous tones has been discussed earlier [12] in more detail. It appeared that for interrupted tones having a sufficiently long duration of single impulses to enable the tone to reach its full loudness value the thresholds were equivalent to those of a continuous tone. In some instances, the interrupted stimuli seemed to yield slightly better thresholds than continuous tones on subjects with little previous testing experience. In general, the audibility of the tone depended more on its duration than on the repetition rate.

Hard of Hearing Subjects.—Many conductively deaf persons have been tested during the last two years with fixed frequency continuous threshold stimulation. It appeared, as stated earlier,[6] that their threshold excursions were quite similar to those of normal persons. In some cases the excursions were large, around 15-20 db.; in others the threshold was very exact and close to the 5 db. limit. The experience of the subjects seemed to influence the thresholds in many cases. Again, perhaps 1 in 20 patients got below the 5 db. line, but not as low as 1 to 3 db. Small excursions were most easily obtained in the higher frequency range and not in the low tones.

As it is obvious that the main interest in the abnormal thresholds lies in the group of perceptively deaf persons, I have confined myself to analyzing testing records obtained in this group. It is also of differential diagnostic interest to see if any differences appear between the recruiting and nonrecruiting types of perceptive deafness.

The recruitment function was determined in each case at the tested frequency by obtaining a point of equal loudness for the compared tones at 20 db. intervals above the threshold of the better ear. With the use of this method, a clear picture of the development of the abnormal loudness increase is obtained and the intensity area where it is most pronounced is observed. The highest intensity obtainable with the audiometer at 1000 cps was 116 db. re 0.0002 microbar.

On the basis of loudness function, perceptive deafness was first divided into a recruiting and a nonrecruiting subgroup. In ordinary clinical material the first category by far exceeds the second in number. From the point of view of threshold studies, the recruiting cases were further subdivided into three categories, viz., asymptotic, straight-line, and delayed recruitment function.[6] We are not concerned with the fourth group observed by Harris, Haines, and Myers [13]—delayed recruitment with asymptotic function—as the emphasis here is on the recruitment function at low levels.

The fourth group studied is that of incomplete recruitment, found in a few cases. It may not make much difference whether the nonrecruiting component of the deafness is a conductive element or a nerve degeneration. However, only those cases of incomplete recruitment were included in which the deafness was entirely perceptive, with equal air- and bone-conduction values, and with an intact conductive mechanism.

The whole perceptive group, 56 patients tested at 101 frequencies, is presented graphically in Figure 3. On the abscissa are the patients with different groups of recruitment function, from asymptotic recruitment to cases with no recruitment. The ordinate shows the average smallest width of the threshold variability obtained during four minutes' recording at one frequency. It will be noted that the majority of the frequencies tested in the asymptotic group show excursions less than 5 db., although there are cases with tracings larger than this limit. The number of greater than 5 db. excursions increases considerably in the straight-line recruitment group, whereas all cases in the delayed group lie above the 5 db. line. In the incomplete recruitment group the tracings are again very small—mostly these cases showed asymptotic growth of loudness near the threshold—and only one case is above the 5 db. line.

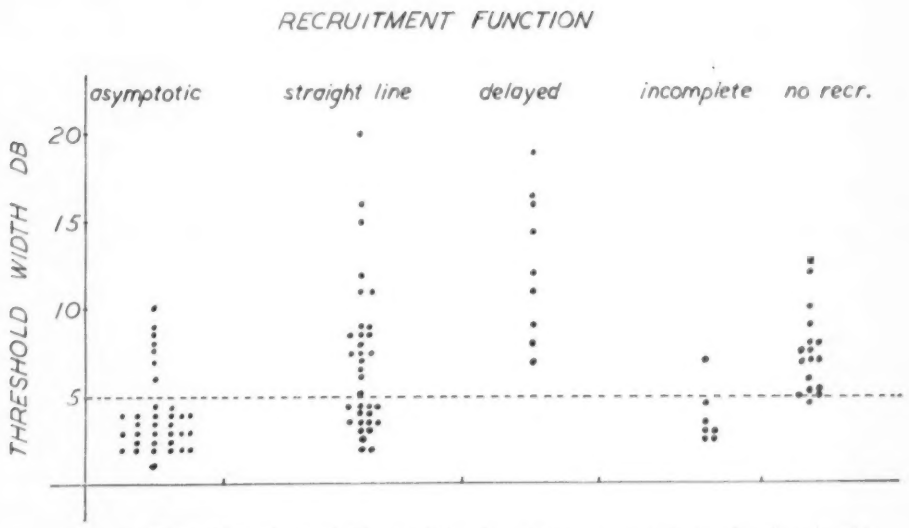

Fig. 3.—The width of threshold variability in relation to recruitment function in the group with perceptive deafness. Audibility increases rapidly in the form of small threshold width with the steepness of recruitment function, but even in the group with asymptotic recruitment there are cases with tracings remaining larger than 5 db.

In nonrecruiting cases there are four instances in which the amplitude is 5 db. or even slightly less, while other cases show larger threshold variability.

In order to get a more detailed idea of the circumstances in the recruiting group, the patients were grouped anew, as shown in Figure 4. Here the abscissa shows vari-

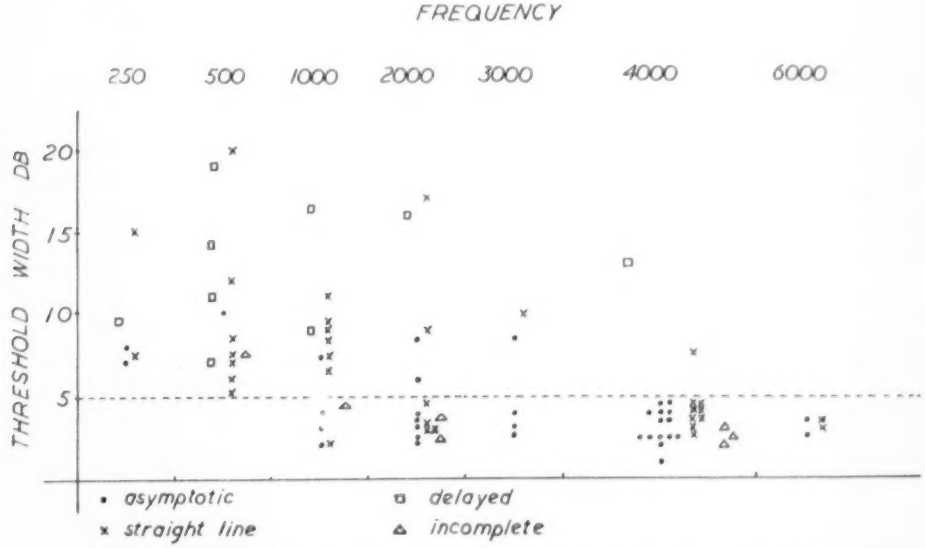

Fig. 4.—The width of threshold variability related to frequency in the various subgroups of recruiting deafness. At the higher frequencies small excursions parallel recruitment, while at 250 and 500 cps all values are 5 db. or larger. The middle frequencies represent an intermediate area.

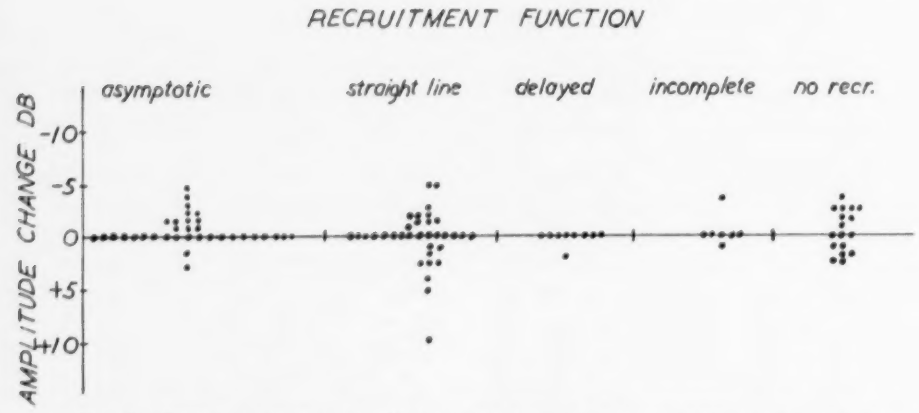

Fig. 5.—Change in threshold amplitude during four minutes' stimulation in the various subdivisions of perceptive deafness. Most of the values show either no change or a change of the order of ±5 db.

ous frequencies from 250 to 6000 cps, while each subgroup is represented by its own mark to indicate the average width of the threshold excursions on the ordinate. The figure shows quite convincingly that in the higher range, 2000 to 6000 cps, the general response in all groups, except that of delayed recruitment, consists in small threshold excursions. The more the frequency is lowered, the greater is the number of cases showing larger than 5 db. variability. At 1000 cps there are mainly asymptotic cases showing less than 5 db. variability, and at 250 and 500 cps all groups showed

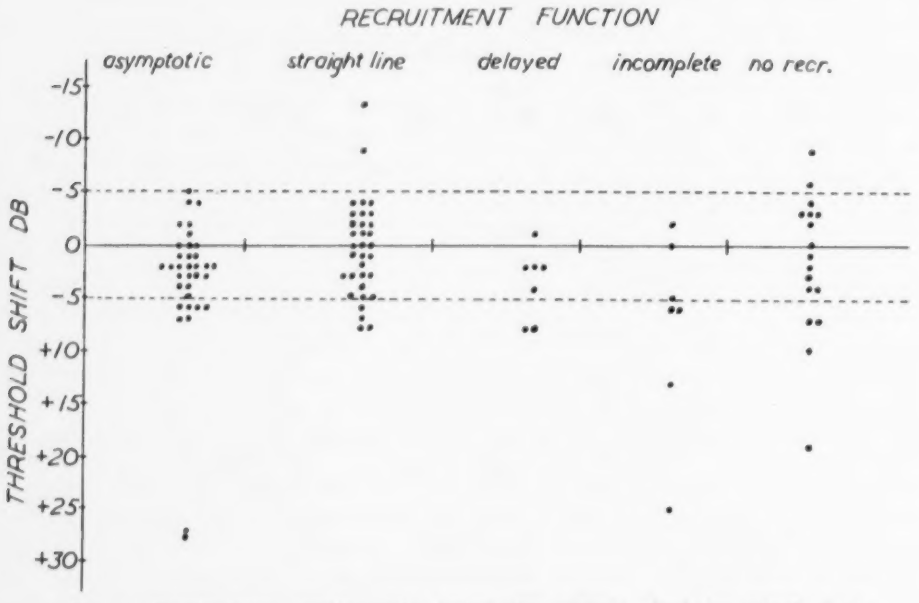

Fig. 6.—Perstimulatory threshold shift during four minutes' stimulation related to recruitment function in perceptive deafness. While most of the values lie within ±5 db., there are a few cases with high fatigability. This phenomenon appears to be independent of the presence of recruitment.

a uniform response of 5 db. or wider excursions. Cases with delayed recruitment are comparable to normal cases, as loudness recruitment is absent at the levels which affect threshold determination.

Threshold Variability as a Function of Time.—In a continuous, fixed frequency threshold recording two different aspects can be observed. One is the variability of the mean individual tracings from minute to minute and the second the variability in the threshold itself, viz., the appearance of possible perstimulatory adaptation at threshold. These two functions in the different groups of perceptive deafness are presented in Figures 5 and 6.

In Figure 5 one can see that a good many cases from the asymptotic and straight-line recruitment groups show diminished excursions in time, a fact that may lead to their being placed into the recruiting category with less than 5 db. excursions. On the other hand, these same groups include some cases in which the excursions increase by a few decibels in time. In the two remaining groups of recruiting deafness there is practically no change, and in the group of no recruitment the values lie within ±5 db. of the initial amplitude.

Figure 6 presents the changes in the threshold itself during four minutes' stimulation. It is seen that most values lie within 5 db. of the initial one-half minute tracing, but there are values deviating from this figure, in some cases even considerably. In a few patients the threshold seems to become better than indicated by the initial recording, and in some cases the sensitivity clearly deteriorates. In the worst instance the threshold after four minutes' stimulation was 28 db. worse than at the beginning of the test. The distribution of the cases with poorer than the original one-half minute threshold seems to be equal in all categories regardless of whether there was recruitment or not.

Comment

Automatic threshold recording at a fixed frequency seemed to yield excursions which on an average varied between 7 and 10 db. This agrees well with the values presented earlier by Lundborg with normally hearing subjects.[3] The limit of 5 db., which was used earlier[6] as a dividing line between recruiting and other patients and which was not passed in Lundborg's[3] normal subjects, seems to be acceptable as the major dividing line. One must always keep in mind, however, that some normally hearing persons, and persons belonging to any nonrecruiting category of deafness, can also give lower than 5 db. threshold excursions. These small values were oftenest seen in the higher frequency range.

As in any subjective testing, there is always a factor of individuality to be reckoned with. Some people seem to be more adaptable to the automatic technique than others, and therefore the normal variation remains quite large.

If one accepts that an average listener with normal hearing or with a conductive hearing loss gives threshold excursions of 5 db. or larger, it remains to be discussed what the incidence of smaller than 5 db. excursions is in the group of recruiting deafness. It was observed earlier,[6] and the finding agrees well with that presented in Figure 3, that most cases with asymptotic recruitment show small tracings but there remain some where the limit of 5 db. is exceeded. More than half the cases in the straight-line recruitment group showed larger than 5 db. amplitudes, and in the delayed recruitment group all were comparable to normal. In the incomplete recruitment group with mainly asymptotic function the excursions were small. In the perceptively deaf cases with no recruitment there were some values on the dividing line, while the majority were larger than 5 db.

The occurrence of small threshold variability seems rather puzzling, but observation of the frequency distribution in Figure 4 will contribute to greater clarity. It

agrees well with Békésy's[2] original observation that it is in the higher frequency range that his new audiometric technique indicates recruitment. It seems that from 2000 cps upward there is a good chance to get small threshold tracings if the patient has a recruiting deafness. Most reliable are tracings at 4000 and 6000 cps, whereas at 1000 cps many of the values are larger than 5 db., and at 250 and 500 cps, regardless of the recruitment function, the width of the tracings tends to be larger than 5 db.

As stated earlier,[5,6] the self-recording technique does not measure the intensity difference limen but is rather a measure of the normal variability of the threshold. Its width depends somewhat upon the technique used, but generally it is between 5 and 15 db. There does not seem to be any appreciable difference in the tracings or tones of various frequencies in normal listeners. For low tones, the audibility may be affected by some secondary factors, such as the subject's pulse rate, breathing, slight movements, and other extraneous noises. On the other hand, the recording at the higher frequencies may be affected by the development of a concomitant high-pitched tinnitus. Nevertheless, there is at present no adequate explanation for the fact that in pathological cases there is a clear frequency difference in the size of the excursions in the self-recorded audiogram.

Finally, it is concluded that, at the higher frequencies, self-recording threshold audiometry can be used as an aid to diagnosis by virtue of its parallelism with the loudness recruitment. One has to keep in mind, however, that all small tracings do not necessarily indicate recruitment but reflect partly a rather large individual variability, and that many cases with recruitment do not show small threshold excursions. This is the case at 1000 cps and especially below it, where the tracings as a rule are 5 db. or larger.

It is obvious that the direct loudness recruitment tests cannot be supplanted by the self-recording technique. The direct tests alone can give precise information concerning the growth of loudness through the whole remaining intensity area of a deafened ear. One can rather say that in those cases in which it is possible to employ neither of the direct tests, owing to a uniform threshold loss, the self-recording technique can help to reveal the rate of the growth of loudness at threshold intensities. If one gets a small threshold tracing it is very likely that the patient would show loudness recruitment also in the direct tests. At higher frequencies this information may be obtainable in nearly all cases, but at lower frequencies larger than 5 db. tracings do not reveal anything concerning either the absence or presence of recruitment.

Testing the threshold at a fixed frequency during some minutes gives the advantage of observing the excursions as a function of time. In some patients, after the initial adjustment, the tracings become smaller and approached their true size. It seems that in many cases the initially larger values were due to inexperience of the patient. On the other hand, in those cases in which the tracings became larger in size than the first one-minute tracing, this was due to the fact that the patients did not quite understand how the test was conducted. This claim is supported by the fact that in all cases with larger than 5 db. increase in amplitude during four minutes' stimulation the absolute threshold became better and moved closer to the threshold measured with the normal audiometric technique. If the larger tracings were an indication of a disappearing recruitment function and fatigue of the ear, as suggested by Hood,[14] the absolute threshold should become worse as a function of time.

When the threshold is measured during extended periods of time, one must allow small variations in the course of the excursions. This was realized by Békésy[2] while demonstrating his new technique, and it is clearly seen in Figure 6, where the distribution around the reference line is mostly

an indication of normal variability. If the variations exceed 5 db. there may be something abnormal in the response.

The deterioration of threshold, lately brought up by Hood,[10] was also seen to occur in some cases (Fig. 6). The values quite agree with those presented by Hood as far as the magnitude of the prestimulatory threshold shift is concerned. In the cases most easily fatigued the threshold shift could be as much as nearly 30 db. However, the frequency of these cases in this material was quite small and the figures for fatigue were generally less than 10 db. It appeared also that in perceptive deafness without recruitment some cases exhibited considerable perstimulatory threshold losses. This finding is in agreement with the earlier papers of Dix and Hood[15] and Reger and Kos[8] and seems to indicate that abnormal fatigability at threshold does not permit differential diagnostic conclusions as to the presence or absence of loudness recruitment. The measurement of perstimulatory threshold shift can, nevertheless, be gratifying, as it gives a clear idea of the ear's functional capacities at low sensation levels.

It may be said in conclusion that, owing to the versatility of the apparatus, the self-recording audiometric technique opens many new approaches to the experimental investigation of the psychophysics of hearing. Some of the characteristics of the response in this type of testing seem at present to be quite well worked out, but there are many others that need careful evaluation. On the other hand, the use of self-recording audiometry is no excuse for omitting the old and proved direct recruitment tests when an evaluation of the recruitment function and an exact analysis of the patient's hearing loss is made.

Summary

A short review is given of papers dealing with self-recording audiometry. Results are presented on 17 normal subjects and on 56 perceptively deaf persons tested at 101 frequencies.

The average normal threshold amplitude is found to vary between 7 and 10 db. Smaller than 5 db. excursions are occasionally seen also in normally hearing persons, and the upper limit only seldom exceeds 20 db. The size of the tracings is similar regardless of whether interrupted or continuous tones are used.

The importance of the determination of loudness function with direct recruitment tests is emphasized. When the amplitude of the threshold tracings is correlated with recruitment function, it is noted that asymptotic recruitment is as a rule associated with small excursions, in the straight-line recruitment function there are many cases with normal tracings, and patients with delayed recruitment show excursions comparable with those of nonrecruiting subjects.

At the higher frequencies, at and above 2000 cps, there is a good correlation between small threshold amplitude and recruitment. At the low frequencies one cannot predict the loudness function on the basis of the threshold tracings.

Normal variability in the threshold amplitude during four minutes' stimulation is of the order of ±5 db. During this testing time the perstimulatory threshold shift may become considerable in a few cases, and it seems to occur in both main subgroups of perceptive deafness.

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REFERENCES

1. Hirsh, I. J.: The Measurement of Hearing, New York, McGraw-Hill Book Company, Inc., 1952, Chap. 4.

2. Békésy, G., von: A New Audiometer, Acta oto-laryng. 35:411, 1947.

3. Lundborg, T.: Diagnostic Problems Concerning Acoustic Tumors, Acta oto-laryng., Supp. 99, p. 1, 1952.

4. Lidén, G.: Loss of Hearing Following Treatment with Dihydrostreptomycin or Streptomycin, Acta oto-laryng. 43:551, 1953.

5. Hirsh, I. J.; Palva, T., and Goodman, A.: Difference Limen and Recruitment, A. M. A. Arch. Otolaryng. 60:525, 1954.

6. Palva, T.: Recruitment Tests at Low Sensation Levels, Laryngoscope 66:1519, 1956.

7. Riesz, R. R.: Differential Sensitivity of the Ear for Pure Tones, Physiol. Rev. 31:867, 1928.

8. Reger, S. N., and Kos, C. M.: Clinical Measurements and Implications of Recruitment, Ann. Otol. Rhin. & Laryng. 61:810, 1952.

9. Lierle, D. M., and Reger, S. N.: Further Studies of Threshold Shifts as Measured with the Békésy-Type Audiometer, Ann. Otol. Rhin. & Laryng. 63:772, 1954.

10. Hood, J. D.: Auditory Fatigue and Adaptation in the Differential Diagnosis of End-Organ Disease, Ann. Otol. Rhin. & Laryng. 64:507, 1955.

11. Palva, T.: Masking in Audiometry, Acta oto-laryng., Supp. 118, p. 156, 1954.

12. Palva, T.: Absolute Thresholds for Continuous and Interrupted Pure Tones, Acta oto-laryng. 46:129, 1956.

13. Harris, J. D.; Haines, H. L., and Myers, C. K.: Loudness Perception for Pure Tones and for Speech, A. M. A. Arch. Otolaryng. 55:107, 1952.

14. Hood, J. D.: Adaptation in Disorders of the Inner Ear and Its Relationship to the Loudness Recruitment Phenomenon, Proc. Fifth Internat. Cong. Otorhinolaryng., Amsterdam, p. 220, 1953.

15. Dix, M. R., and Hood, J. D.: Modern Developments in Pure Tone Audiometry and Their Application to the Clinical Diagnosis of End-Organ Deafness, J. Laryng. & Otol. 67:343, 1953.

Case Reports

Bilateral Cricoarytenoid Ankylosis

Surgical Treatment

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Cricoarytenoid ankylosis was first called to my attention by the excellent article of Montgomery, Perone, and Schall on that subject. At the time of its publication, I had just seen a case of bilateral abductor paralysis of the larynx of unknown etiology. A review of the clinical findings revealed my diagnostic error.

The diagnosis of bilateral cricoarytenoid ankylosis may be made upon the finding of the following symptoms and signs: (1) A history of dyspnea on inspiration and expiration which is more marked on inspiration. (2) Minimal voice impairment. (3) Mirror laryngoscopy revealing the arytenoids fixed in the mid-line with a typical bowing of the anterior two-thirds on inspiration, as reported by Montgomery et al. (4) Confirmation with direct laryngoscopy showing the arytenoids to be immobile and producing pain on attempting to move them.

The differential diagnosis involves the various causes of bilateral abductor paralysis, and as these are eliminated we virtually rule in a bilateral cricoarytenoid ankylosis. A consideration of these diseases from the standpoint of their pathologic physiology brings up an important difference in their surgical treatment and end-result. The basic facts to be kept in mind are that in one we have a permanent loss of muscle function and in the other the laryngeal muscles are temporarily impaired in their action due to immobile arytenoids.

Normally, the arytenoids act as a shifting fulcrum about which the intrinsic musculature of the larynx acts. Fixation of this fulcrum alters the action of the laryngeal muscles. At this time it is reasonable to believe that only the thyroarytenoideus can function and that it produces the typical inspiratory bowing. The minimal airway thus provided obviates the emergency tracheotomy necessary in bilateral abductor paralysis.

Arytenoidectomy alters the action of the laryngeal muscles which no longer have a fulcrum to fix against. During inspiration under their new status, it is my belief that the thyroarytenoideus and the lateral cricoarytenoideus contract to cause a further exaggerated bowing of the vocal cords no longer limited to the anterior two-thirds of the cords, while their opposites relax. On the other hand, on phonation the posterior cricoarytenoideus and the interarytenoideus contract and the thyroarytenoideus and lateral cricoarytenoideus relax to allow a complete closure of the glottic chink.

In support of the above, I believe that we can accept the findings, teachings, and results of Sister Kenny as applied in the treatment of poliomyelitis. Postparalytic polio patients have had muscles trained to assume functions that were formerly alien to them. Here we have a comparable situation in which muscles forget their action by virtue of forced inactivity, and when released once again the natural instinct of an organism to survive compels the muscles to assume new functions.

I propose that the treatment for bilateral cricoarytenoid ankylosis consist of a simple bilateral arytenoidectomy approached by an anterior commissure laryngofissure preceded by an orderly tracheotomy. This entire procedure may be done under general anesthesia if the anesthetist, using local anesthesia,

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first inserts a small intubation tube. The technique is the same as described by Montgomery et al. with the exception that the vocal cords are not sutured laterally to the thyroid cartilage. The incision of the cordal mucosa is closed with 000 chromic absorbable surgical (gut) sutures. Convalescence from such a procedure is rapid. With both arytenoids removed, it should be possible to decannulate the patient within two weeks.

Report of Case

A 66-year-old white man was first seen by me on Feb. 3, 1956, because of a severe acute laryngitis with dyspnea. Treatment had been initiated the preceding night by another physician, and the patient recovered satisfactorily from this episode. A history of intermittent dyspnea of four year's duration was elicited. This had become more or less constant during the past two or three years and was worse with colds and somewhat more marked on inspiration. There was no history of severe laryngitis, neck trauma, or arthritis; although he did recall being hoarse on two occasions in the past, for which no medical attention was required. Previous illnesses consisted of pneumonia 40 years ago, a duodenal ulcer intermittently under treatment since 1937, cataract surgery in 1943 and 1946, and a bilateral hernioplasty in 1949. In addition, in 1955 he had a complete medical study and was hospitalized for several months because of a suspected tuberculosis with a discharge diagnosis of pulmonary fibrosis and emphysema.

Initial examination was negative except for a small polyp in the left middle meatus and an acutely inflammed larynx with a small glottic chink which when the inflammation subsided was estimated to be 2 mm. in width on inspiration. On March 15, under local anesthesia, a direct laryngoscopy and cervical esophagoscopy were done to confirm the diagnosis of cricoarytenoid ankylosis and to rule out malignancy. At this time, the typical inspiratory bowing was again noted, and pain was produced on attempting to displace the arytenoids laterally. A unilateral arytenoidectomy with a temporary tracheotomy was advised.

On March 30, after the patient was intubated under local anesthesia, a general anesthetic was given. A mid-line incision was made, and progressively a tracheotomy, mid-line thyrotomy, and right arytenoidectomy were done. A vertical incision was made over the right arytenoid which was then submucously dissected free. No cleavage plane could be found at the cricoarytenoid articulation, where it was necessary to use the sharp

Right arytenoid cartilage showing the spiculated base where it was adherent to the cricoid cartilage.

Freer septal elevator to separate the cartilages. The mucosal incision was closed with interruped 000 chromic absorbable surgical (gut) sutures. At this point, because of the current popularity of stapes mobilization, I and my colleague, Dr. James Clopton, tried to manually mobilize the left arytenoid without success. The wound was then closed in layers.

The patient was discharged home on the sixth postoperative day. The tracheotomy tube was partially corked on the 11th postoperative day, completely corked on the 17th and removed on the 28th day. I estimate his present airway to be approximately 4 mm. in the mid-glottis and 2 to 2½ mm. in the posterior glottis. Subjectively, the patient states that there is no change in his voice and no shortness of breath. Gauging dyspnea by one's ability to walk, he stated that he had to rest after walking two to three blocks; however, further inquiry revealed that this was because his back hurt. It is felt that decannulation would have been possible much sooner had both arytenoids been removed.

Summary

The diagnosis, differential diagnosis, pathology, and treatment of bilateral cricoarytenoid ankylosis are discussed. The surgical treatment of direct simple bilateral

arytenoidectomy is advocated as giving a good voice and the best possible airway for this condition. A case is presented to illustrate the reasoning back of this concept.

1308 N. Broadway.

REFERENCE

1. Montgomery, W. W.; Perone, P. M., and Schall, L. A.: Arthritis of the Cricoarytenoid Joint, Ann. Otol. Rhin. & Laryng. 64:1025, (Dec.) 1955.

Clinical Notes, New Instruments and Techniques

Nasolaryngoscope

LOUIS K. PITMAN, M.D., New York

This is a preliminary report on the examination of the larynx and surrounding areas with the aid of a new instrument and new approach.

The present methods for viewing the interior of the larynx are through the mouth, by indirect or direct laryngoscopy.

These methods have their limitations, i. e.:

(*a*) Marked sensitivity of the throat, causing gagging.

(*b*) Anatomic difficulties.

(*c*) The inconvenience of inserting a straight tube over the dorsum of the tongue and lifting the epiglottis, to view the larynx.

(*d*) Examination requires the aid of a specialist.

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From the Department of Surgery, Head and Neck Service, Albert Einstein College of Medicine, Yeshiva University, and Bronx Municipal Hospital Center.

My new approach is through the nasal passage, entering the nasopharynx. It uses a scope of suitable caliber and magnification. The total magnification required depends upon the choice of a suitable, objective-tube lens-eye-piece combination. The sighting prism, viewer, or objective is located at the tip of the scope so that when the scope is in position the soft palate and uvula will not obstruct the view of the larynx. A better view is obtained by having the patient project the tongue forward during visualization. The illumination should also not be obstructed; therefore, the illumination leaving the scope must be adjacent to the viewing prism. In other words, the objective prism and the rays of light leaving the scope must be confined to the tip of the instrument, within a space not exceeding 3/8 in., for the space between the uvula and the posterior wall of the pharynx is limited.

Various types of illumination have been attempted:

(*a*) A condensing lamp fixed into the tip of the scope and at a right angle to its axis.

(*b*) A condensing lamp above or below the sighting prism, which directs the rays of light downward.

(*c*) A right-angle type of lamp, overhanging the tip of the scope.

(*d*) A light enclosed in a tube and its forward rays bent downward by a reflector, without a prism.

The above did not meet the requirements of the sharp concentrated illumination necessary. I chose the prism-type illumination, as shown.

To the long axis of the scope, a light carrier is attached, with the aid of a snap-on sleeve. At the terminal end of the carrier, there is a deflecting prism (type used in May ophthalmoscope but of smaller dimensions). Before the prism there is a condensing lens which concentrates and regulates the light rays. A light on a carrier is introduced into the lumen of the carrier, which can be moved forward and backward for maximum illumination.

Method of Use

Select the desired nasal passage and, after cocainizing and shrinking the mucosa, introduce along the floor of the nose the assembled instrument adjusted to downward vision and illumination. Have the patient protrude the tongue. The larynx is now ready for visualization. The examination is done at leisure and without any discomfort to the patient.

1749 Grand Concourse (53).

Modified Jackson Laryngoscope

C. J. HEINBERG, M.D., Pensacola, Fla.

The modified Jackson laryngoscope combines the Lewy self-retaining bar, the Jackson handle, and a new blade for better visualization of the larynx and greater manipulation of instruments in the approach to the larynx. By exposing the larynx this instrument, with the self-retaining qualities of the Lewy bar, makes possible angulation of the forceps and better vision of the procedure being done. After using the instrument, Dr. Francis LeJeune, of New Orleans, stated, "It is the closest thing to suspension that I have run into, and I think it is excellent."

Accepted for publication Dec. 17, 1956.

109 N. Baylen St.

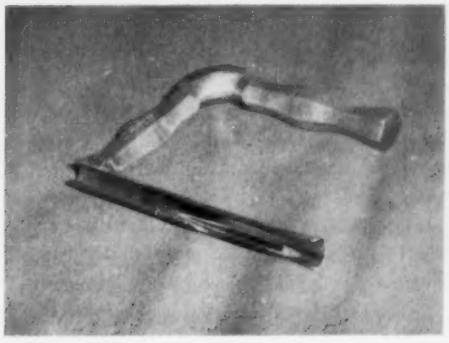

Fig. 1.—The modified Jackson laryngoscope with wide-open blade for better visualization of larynx and opening on side for ease of instrumentation.

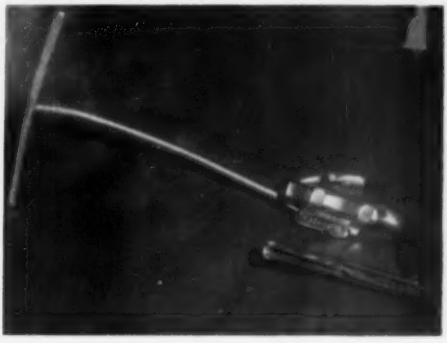

Fig. 2.—The instrument with Lewy bar attached for self-retaining visualization of the larynx, allowing both hands to be free for instrumentation.

Progress in Otolaryngology

Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Internal Ear

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1952

BRUCE PROCTOR, M.D., Detroit; EMERY PICK, M.D., Los Angeles, and EUGENE L. DERLACKI, M.D., Chicago

The literature on chronic progressive deafness has become so extensive that it necessitates for abstracting purposes brevity and the omission of repetitive reports. The papers reviewed are arranged under the headings of otosclerosis, Ménière's disease, toxic labyrinthitis, acoustic trauma, function and diseases of the labyrinth, hearing tests and auditory training, and miscellaneous contributions on deafness.

Otosclerosis

Hall [1] studied the bone removed from 120 fenestration operations, correlating the clinical data with the pathology. Various stages of the disease were demonstrated. These changes seemed to center around the vascular spaces, thus supporting the vascular theory of the origin of otosclerosis.

The Nylens [2] suggest that a particular vascularization of the otic capsule and/or a metabolic disorder, mainly due to imbalance of the internal glands of secretion, are of importance in the genesis of otosclerosis. At present the most plausible disturbance seems to be a slight primary or secondary dysfunction of the parathyroid glands influencing the metabolism, especially of enzymes and mineral salts.

In otosclerosis there are preotosclerotic and otosclerotic changes (Brunner [3]). The preotosclerotic changes consist of (1) an overproduction of both precollangenous substance and primitive bone and (2) an extensive degeneration of the cartilage at the frame of the windows. These changes are hereditary and do not produce clinical symptoms. They are called "preotosclerotic" because (1) they are frequently associated with actual otosclerosis, (2) they occur also in instances without actual otosclerosis, (3) they are not associated with other diseases of the bone of the labyrinth, and (4) they are, as far as the "blue mantles" are concerned, distributed all over the petrous bone. Brunner considers the actual otosclerotic nidus as a genuine tumor of the bone of the labyrinth capsule.

Asherson [4] has noted that the onset of otosclerosis coincides with the period in which ossification sets in in the epiphyses of long bones and correlates this with an observation that an attack of otitis media in childhood is almost always a guarantee against the development later of clinical otosclerosis. His differential diagnosis of otosclerosis includes adhesive otitis media with fixation of the stapes footplate, primary ankylosis of the stapes footplate due to trauma (dislocation produced by head injury, blast, or local trauma to middle ear), ankylosis of ossicular chain, fixation of stapes from within the labyrinth (hydrops, tumor), and permanent stapedius spasm.

Cabanettes [5] distinguishes three types of atypical otosclerosis: (1) unilateral type,

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characterized by a triad of unilateral predominantly conductive deafness, tinnitus, and vertigo; vertigo, which almost never occurs in the bilateral form, is a constant and severe symptom and is markedly associated with changes in the position of the head; in some cases bilateral otosclerosis develops; (2) the type associated with chronic adhesive otitis media; (3) cochlear forms with the otosclerotic focus affecting other regions of the labyrinthine capsule while sparing the oval window. In this cochlear form the tinnitus is of medium pitch and the whole tonal range shows marked hearing loss from the onset, whereas in other cases of labyrinthine deafness of peripheral origin (e. g., toxic cases) there is a predilection for the high frequencies.

Cawthorne[6] reviews the clinical picture of otosclerosis and presents observations made on 1150 cases of otosclerosis which were drawn from a consecutive series of 2385 patients complaining of deafness. The incidence in females was 63% and in males 37%. Hearing loss was aggravated by pregnancy in 61%. A family history of deafness was elicited in 611 cases. Dizziness of a slight degree was noted in 262 cases and of a severe degree in 16 cases. Schwartz's sign was found in 10%. Cawthorne reports a noticeable increase in the proportion of fair-haired otosclerotic subjects over the normal population.

Rosen[7] reflects the skin up from the depth of the external canal together with the posterior portion of the tympanic membrane to allow access to the region of the stapes and oval window. The degree of stapes fixation can be determined. It is a useful method in checking diagnosis in doubtful cases. It is useful as a means of mobilizing the stapes to improve the hearing. If adhesions, etc., are present instead of otosclerosis, they can be dealt with and an unnecessary fenestration avoided.

Fenestration is useful even when the goal of hearing the spoken word at a distance of 3 ft. postoperatively cannot be attained. Hahlbrock[8] enumerates (1) selected patients who are for all practical purposes deaf but where it is hoped that the operation may recover perception of sound which will facilitate orientation in space and (2) patients who are for all practical purposes deaf but where it may be hoped that enough hearing can be recovered so that by use of a hearing aid auditory function can be restored to the point where the patient is able to communicate with others.

Bone-conduction audiometry under proper test conditions is as reliable as air-conduction audiometry (Shambaugh[9]). The bone-conduction audiogram in pure stapes ankylosis shows a notching that is partially reversible by fenestration. This notching is believed to be due to the mechanical effect of stapes ankylosis. The actual cochlear nerve response of a patient with otosclerosis is determined by subtracting the mechanical otosclerotic notch from the bone-conduction audiogram. The air-bone gap measures the conductive component of the hearing loss (after correcting for the otosclerotic notch). The maximum conductive hearing loss for stapes ankylosis is 55-60 db. for the speech frequencies. Fenestration leaves an unrestored residue of conductive loss of 20-30 db. The probable result of a successful operation may be calculated by drawing a curve of 25 db. below the corrected bone-conduction curve. The probable gain from a successful operation depends on the air-bone gap in that case. The speech reception and discrimination tests are useful in confirming and supplementing the information obtained from the pure-tone air and bone audiograms.

The fenestra should be made with the least trauma and manipulation, for the flatter and closer the apposition of the flap to the opening the better (Hall[10]). The flap should contact the membranous canal. For controlling blood pressure and hence the hemostasis Hall uses the parasympathetic block method with the spinal anesthesia carried to the level of the first dorsal segment. Cases need not be rejected because the maximum gain must fall short of social adequacy. The fenestrator must be prepared to perform revisions with a reasonable hope of success (80%). In revisions a small

canal fenestration can be made behind or in front of the previous site so as to expose the membranous canal. Any traction on the canal by adhesions can be immediately recognized.

The usual causes for failure in fenestration are osteogenic closure and postoperative serous labyrinthitis (Shambaugh [11]). Experimental studies suggest six basic factors that control osteogenesis after fenestration:

1. Removal of all particles of bone from the fenestra.
2. Enchondralization of the fenestra.
3. Covering the fenestra with a tightly fitting skin flap.
4. Endosteum hemming of the fenestra.
5. Burnishing the bone around the fenestra.
6. Prevention of fibrosis in the fenestra.

Experimental studies and clinical observations suggest three basic factors concerned with the production of postoperative serous labyrinthitis: (1) hemorrhage into the labyrinth, (2) sterile traumatic inflammation of the plastic flap over the fenestra, and (3) infection.

Closure of the fenestra is due to the leakage of serum and blood from the flap or bleeding from the unsealed endosteal vessels (Hall [12]). This probably forms a coagulum under the flap, and in this coagulum the fibrous tissue is produced. The fibrous tissue goes on eventually to bone formation. In 16 revisions more than 18 months old he obtained 11 successes. Hall prefers to wait two years before performing a revision. Persistent discharge above the flap may be treated by the elimination of nasopharyngeal sepsis and the application of trichloroacetic acid to the discharging fistula. Necrosis of bone or cartilage may be responsible for the persisting discharge. Trauma from the retractor may be responsible for the latter. Hall recommends secondary skin grafting five to seven days after fenestration.

Rüedi et al.[13] perform a two-stage fenestration operation. The first stage is done with a combined endaural and retroaural approach. The incus is removed. The tympanomeatal flap is placed over the unopened horizontal canal. A skin graft is placed over the exposed bone in the mastoid cavity. A few months later the cavity is reopened, the skin elevated from the horizontal canal, and fenestration performed. The skin flap is replaced over the fenestra, fitting perfectly. The fenestration is done in a completely dry field. The reported results were very good.

Klotz [14] describes Sourdille's two-stage operation using the posterior approach. The incus is preserved, and the fistula is placed on the superior surface of the horizontal canal. The mastoid cells are completely exenterated. The head of the malleus is removed. Audiograms and statistical results of the surgery were not given.

Garson [15] describes a closed method of fenestration which he has used in 35 cases. The postaural approach is used, the incus removed, and the fenestrum created. A free conjunctival graft is used to cover the fenestrum. It is in turn covered with gold leaf and then packed. The incision is left open. A week later the dressings and the gold leaf are removed and the incision closed with capillary drain, which is left in position for four or five days. By this method improvement in hearing is obtained, convalescence is short, and the risk of a chronically discharging ear is almost eliminated.

A minimum of pneumatic cells are removed from the mastoid during fenestration operation in order to promote fast healing (Loebell [16]). The cells of the perilabyrinth and of the epitympanum are eradicated whenever feasible, in order to reduce the possibility of a postoperative infection in close proximity to the new window. Only 3 out of 200 cases required subsequent evacuation of persistently suppurating cells. In these cases it was difficult to reestablish an epithelial lining and postoperative treatment was prolonged.

Vyslonzil [17] finds that controlled hypotension facilitates the fenestration operation. In patients over 50 years of age initial pressure of 165/85 was reduced to 60/45; in younger patients the level maintained was 55/40. Once the desired level has been established through medication it is maintained by means of correct positioning of the patient. Hypo-

tension for a period of 40-60 minutes does not result in permanent damage to the circulatory system. Hypotension is induced when the surgeon is ready to create the fenestra. Better vision contributes to greater exactness. Formation of capillary thrombi keeps the region postoperatively anemic. It is necessary to increase the blood pressure gradually so as to protect these thrombi and prevent postoperative hemorrhages into the labyrinth. Blood pressure in the cerebral vessels is carefully checked by measurement of the pressure of the arteria centralis retinae. The levels necessary to facilitate the operation never resulted in trophic disturbances of the brain.

Because of the theoretical similarity between otosclerosis and joint fixation in arthritis Goodhill [18] treated various types of otosclerosis with oral cortisone. In none of the 16 cases of otosclerosis so treated did cortisone improve the hearing. Temporary subjective improvements due to elation and euphoria were not confirmed audiometrically and did not persist.

Rambo [19] administered intramuscularly 10 mg. of cortisone daily to two fenestrated Rhesus monkeys for 25 days. Ten months later the animals were killed and the temporal bones studied. In each case the fenestra had closed as a result of new bone formation. It is concluded that cortisone has no inhibitory effect on osteogenesis.

Lansberg [20] feels that corticotropin (ACTH) may have some value in fenestration surgery to prevent postoperative wound reaction, especially serous labyrinthitis. Rabbits showed an inhibited wound reaction in corticotropin-treated animals after skull trauma. Because a high concentration of cortisone must be administered to obtain the desired catabolic effect, the author favors the local application of this hormone.

Riskaer [21] treated nine otosclerotic patients who had had fenestration 9-30 months previously without maintained improvement in hearing. They were given corticotropin in doses corresponding to 20-30 mg. intramuscularly four times daily for eight days. In all nine cases the treatment had no effect whatever, either on the ears not operated on or on the fenestrated ears. One otosclerotic patient whose hearing had been impaired for only three months before the treatment obtained considerable improvement, a fact which seems to suggest that fresh cases may respond favorably. The treatment had no unpleasant side-effects, such as acoustic or vestibular reaction, in spite of considerable fluid retention.

Shambaugh [22] presents histological evidence supporting Siebenmann's hypothesis that the cochlear nerve degeneration of otosclerosis is due to the diffusion of toxic products from the pathologic bone where the focus reaches the endosteum of the cochlea. Increased cochlear nerve degeneration does not occur in the great majority of cases following fenestration. When it does occur it is commoner in the ear not operated on. Partial loss of regained hearing occurred in about one-fifth of the cases followed for 5-10 years after fenestration (osteogenic narrowing of the fenestra, postoperative hydrops, cochlear nerve degeneration). The majority (70%) of fenestrated cases followed 5-10 years after surgery maintained the level of hearing attained at the one-year postoperative test.

Both the final level of hearing and the decibels gained are significant and important in assessing the results of fenestration surgery (Shambaugh [23]). With the improved fenestration operation 75%-80% of otosclerotics without nerve involvement for speech frequencies (A), 40% of cases with early cochlear nerve degeneration for 2048 (B), and 10% of cases with cochlear nerve involvement for two or all three of the speech frequencies (C) reached the level of practical hearing at the end of one year. In terms of decibels gained or lost, 94% of A Cases, 90% of B Cases, and 75% of C Cases showed a significant hearing gain at the end of one year. The hearing after operation equaled the preoperative hearing with the hearing aid for speech reception and surpassed the aid for discrimination.

House [24] reported on the long-term results of fenestration surgery. Of 200 patients on whom such surgery was done at least five years ago, 44% could be followed. In these cases complete records with questionnaires and audiograms were obtained. Fifty-five per cent of patients have less than 30 db. loss two years after surgery. Thirty-four per cent have less than 30 db. loss at the end of five years. Sixty-five per cent are pleased with the hearing result, and fifty-six per cent do not feel the need of a hearing aid five years postoperatively. In 1056 two-year cases 54% showed practical hearing. Discharge was present in the ear operated on in 14% of cases, but all cavities had been dry at some time during the five years. The better the nerve function preoperatively the better the end-result, and the better the final hearing result the less tendency toward deterioration. Deterioration seems less common in the fenestrated ear than in the opposite intact ear. The two greatest causes of failure in obtained permanent hearing gain are improper selection of patients for surgery and the predestined progressive deterioration of nerve function encountered in otosclerosis.

Under certain conditions the fenestra may transmit air-borne sounds to the cochlea (Skoog [25]). In many fenestrated cases with strong fistula response there may occur an interference between the sound entering the labyrinth via the fenestra and sound waves entering via the round window. By covering the drum this interference may be diminished and the hearing could be improved. In cases with a weak fistula response there is fibrosis in the fistula with only slight interference for sound, so that when the drum is blocked the hearing is not improved or may even be made worse.

Nilsson [26] made extensive studies in 55 cases on the changes in pure-tone transmission with fenestration. Some mechanical aspects of the middle ear are discussed, namely, the middle-ear impedance matching effect, the application of the impedance formula, and the role of the round window. Following fenestration the predominant stimulus reaches the inner ear via the new window. The experience of hearing improvement postoperatively after blocking of the drum membrane lends added weight to this assumption.

Nilsson performed various tests on the subjects. Test I was done the day before operation, Test II immediately before operation, Test III immediately before fenestration, Test IV immediately after fenestration, and Tests V-VIII were done in connection with fenestration in order to study the effect of various types of blocking on sound transmission. Test IX was done in 10 cases three to six months after operation. Comparison of results in Tests I and II showed no significant difference, indicating that premedication had no effect on threshold determinations. Test III showed a rise of 2-3 db. in the air-conduction threshold at frequencies below 1000 cps and an even greater one at the higher frequencies (15 db. at 8000 cps). This impairment of hearing in Test III is ascribed to altered conditions of transmission, mainly through the flap having been placed in such a way as to block the pathway to the round window. In Test IV an improvement of both air conduction and bone conduction was obtained in all cases of clinical otosclerosis except two. The negative results were assumed to have been due to otosclerotic occlusion of the round window. The air-conduction curve in Test IX showed a fall for the lower frequencies in relation to the immediate results. This finding is attributed to impaired conditions of transmission mainly due to covering of the fenestra by the flap. In Test IV supernormal bone conduction was demonstrated at the lower frequencies, which may have been due to abnormally good mobility of the perilymph.

McKenzie [27] considers an increase in deafness during pregnancy as an urgent indication for operation.

Even though a fenestration has been correctly performed and a well-functioning window created, an unsatisfactory result may occur due to traumatic inflammation of the mobilized flap and the inner ear (Thullen [28]).

More frequently hearing loss is due to disturbances originating from the tympanic membrane, middle-ear cavity, or auditory tube. Perforation of the tympanic membrane during or following the operation results in deterioration of hearing, which can be corrected by a prosthesis. Too little attention has as yet been given to interference with tubal function.

With regard to the tympanic membrane, its function depends not only on its integrity but also on such factors as tension, shape, and volume. A cotton plug placed against the tympanic membrane often results in improved hearing.

Zangemeister [29] states that hearing in the contralateral ear not operated on is occasionally improved after the usual type of operation (horizontal canal), but the improvement occurs more frequently and to a higher degree with endocranial fenestration (vertical portion of the superior canal). In this latter instance hearing of the spoken word as well as audiographic tests may reach the values of the ear operated on. Clinical and experimental studies show that improved hearing in the ear not operated on is simulated by errors in measurements, which can hardly be avoided and are due to bone conduction. This explains the finding that hearing in the ear not operated on is particularly improved in cases in which there is a marked postoperative increase of bone conduction.

Failure of a fenestration operation may be defined as a result that falls short of the anticipated hearing improvement, as estimated from the preoperative tests (Shambaugh [30]). The most important cause for failure at the present time is postoperative serous labyrinthitis due to bleeding or infection. Labyrinthine hydrops and increased cochlear nerve degeneration are occasional unavoidable causes for failure.

The causes of closure are osteogenesis, fibrosis, and labyrinthitis (Meltzer [31]). Labyrinthitis of a varying degree results from the surgery itself. Blood beneath the flap becomes the matrix for fibrosis. The ideal case for revision is one that obtained good improvement postoperatively for four to six months and then gradually returned to the preoperative level. Revision should not be done before the end of the first postoperative year. A rapid depreciation of the initial improvement usually indicates fibrosis. Increasing experience of revisions suggests that 25%-30% may reach the practical level and perhaps another 10%-15% may attain some improvement.

Meurman and Savolainen [32] upon examining 63 discharging fenestration cavities found that the predominant organism in the primary cultures was Micrococcus—pyogenes var. aureus in 19 cases, Pseudomonas aeruginosa in 21 cases, and Corynebacterium pseudodiphtheriticum in 7 cases. Bacteriological studies were made of the auditory canals of 100 healthy adults and of the nose, pharynx, and feces of some of the patients before fenestration in an attempt to determine the source of the infection. Since strains of P. aeruginosa were not found, they concluded that the infections enter postoperatively from outside sources.

Passow [33] observed 40 cases of vestibular disturbances immediately following fenestration for a period of 10 weeks to 20 months. In 30 patients vestibular complaints disappeared within eight weeks. Spontaneous nystagmus lasted for 1-14 days postoperatively, while vibratory nystagmus persisted as a rule for more than four months, and positional nystagmus up to eight months. Experimental stimulation of the vestibular apparatus, repeated at weekly intervals, showed that in the majority of patients the response was considerably reduced on the side operated on as compared with that of the other; five months postoperatively the caloric response became bilaterally equal. The rotatory response was on the average bilaterally equal nine weeks postoperatively. On the whole vestibular disturbances are so much diminished within eight weeks postoperatively that they are no longer registered as interferences.

Meurman and Pursiainen [34] studied the preoperative caloric and tuning tests and did not find a weaker reaction in ears with more defective hearing on which fenestration

operation was later performed. After fenestration operation the fenestrated ear responded more weakly to the caloric stimulation than the nonfenestrated ear. Likewise the fenestrated ear did not react as readily to the caloric test as the nonfenestrated ear. In the fenestrated ear the average duration of nystagmus was greatly reduced. In the tuning tests the average duration of the nystagmus to the fenestrated ear was shorter.

Huizinga [35] studied the sound reaction of Tullio in the pigeon and the fenestration operation in man. The microphonic effects of sound upon the crista in pigeons can be obtained if an opening is made in the bony semicircular canal. This effect closely resembles the cochlear effect. The Tullio reaction to sound becomes possible so that vestibular reactions appear. The existence of this microphonic effect on the crista proves that in Tullio's reaction the crista vibrates synchronously with the frequency presented to the ear. The Tullio reaction is brought about by the fact that by the new opening movements of the inner-ear fluid are produced in the vestibular part of the labyrinth. The cupula moves to and fro. These movements do not neutralize each other owing to the one-sided sensitivity of the ampulla (Ewald's law). Cumulation causes strong reactions in one direction, just as though an endolymph current took place, which, however, is not the case, as can be proved by plugging the canal. In the pigeon Tullio's reaction causes strong vestibular responses. In man after fenestration very little reaction to sound is found, due to a high threshold to Tullio's reaction. This is due to several factors: (1) After fenestration the irritability of the labyrinth is diminished, and (2) the fenestra is covered with the skin flap. No reaction was seen at 115 db. in a number of patients with good results after fenestration though the fistula test always gave positive results. The vestibular symptoms after fenestration are not necessarily due to labyrinthitis. The severity of symptoms depends upon the quantity of perilymph fluid which escaped. This results in vestibular symptoms to the homolateral side (nystagmus to the opposite side).

If an aperture is made in a pigeon's osseous semicircular canal, sound waves at levels above 70 db. give a distinct vestibular reaction due to stimulation of the crista (Huizinga [36]). When the columella is disrupted the threshold rises to 100 db. This 30 db. difference is comparable to the limits of improvement of fenestration in man. With successful fenestration the Tullio reaction should only rise at intensities exceeding 100 db. The fenestration almost certainly impairs function of the crista. This factor plus the presence of a skin flap over the fenestra raises the threshold even higher, so that humans have shown no Tullio reaction with stimuli as high as 115 db. Hence patients who have had successful fenestrations should very seldom be subjected to troublesome vestibular discomfort.

Labyrinthine Hydrops

Lempert et al.[37] present a new interpretation of the histopathologic findings in the labyrinths of cases of Ménière's disease. Lempert's group does not believe that distention of the cochlear duct and absence of the normal soft perisaccular connective tissue surrounding the saccus endolymphaticus are sufficient evidence that Ménière's symptom complex is caused by either hypersecretion or poor resorption of endolymph. They feel that this postmortem cochlear pathology and the symptoms therefrom may be the sequelae of what is primarily a disease of the vestibular labyrinth. The membranous external semicircular canal with its ampulla and in six cases part of the utricle comprised the specimens for study. "Maculae from four patients and a crista from two were observed . . . all showed marked degenerative changes of a chronic type." The inner lining of the six membranous external semicircular canals was invariably found to be studded with vesiculated epithelial excrescences varying in size and number, suggestive of the formation of epithelial fluid collections. They believe that the epithelial vesiculation

of the inner lining of the vestibular membranous labyrinth is always pathological, whether or not it is accompanied by Ménière's symptom complex. The authors conclude the Ménière's disease is a chronic progressive "herpetic neuritis" of the vestibular labyrinth of either toxic or trophic origin. Furthermore, they theorize that a Ménière's attack is due to periodic rupture of continuously forming vesicles with the release of a toxic fluid, chemically different from normal endolymph, and that all the symptoms of Ménière's disease, as well as the cochlear endolymphatic distention, can be explained as a result of the contamination of and toxic effect upon the endolymph by this vesicular fluid.

Furstenberg [38] wishes to keep the clinical signs and symptoms of Ménière's disease as a well-defined entity explainable as responses to stimuli originating within the vestibular labyrinth and traveling along definite anatomical pathways to the vestibular nuclei, with subsequent central connections with such nuclei as the abducens and oculomotor or the dorsal efferent nucleus of X.

This well-defined symptomatology consists of an attack of vertigo with nausea and vomiting in the severe seizures, a spontaneous nystagmus of varying degrees, a perceptive type of hearing impairment in the ear to which the vertigo is referred, and a tinnitus which is frequently aggravated immediately preceding and during the attack. The author finds reason to question the relationship between Ménière's disease and those certain signs and symptoms, sometimes reported in the literature as integral parts of Ménière's disease, when the anatomical considerations are incongruous. For instance, when vertigo dominates the clinical picture but is associated with a burning sensation in the pharynx (ninth nerve), numbness on one side of the face and ear, or a typical tic douloureux (fifth, seventh, ninth, and tenth nerves), it is impossible to reconcile these findings with an isolated lesion in the internal ear, and their presence is an indication for study of other regions of the central nervous system.

Wildhagen [39] reports 10 cases of typical Ménière's syndrome with, in general, insignificant finding in the cochlear and vestibular examinations. X-rays of the cervical vertebrae revealed deformities and some degree of obstruction of the intervertebral foramina on one side in all cases. The patients had a corresponding spinal or paravertebral tenderness. In these cases the causes of Ménière's syndrome were probably due to vascular disturbances in the distribution area of the vertebral artery among nerve bundles. The treatment was extension of the cervical part of the vertebral column by Schanz' method.

According to Myer zum Gottesberge,[40] the audiogram in Ménière's disease is characteristic. The loss of hearing is in most cases equal for all frequencies: A slow fall or rise of the curve in the higher frequencies occurs. In 90% of cases there is a pronounced difference between the sides: In one ear hearing is either normal (50%) or only slightly diminished (40%). The hearing in the other ear is usually severely impaired. Recruitment is generally present.

The psychosomatic aspects of Ménière's disease were studied by Fowler and Zeckel.[41] Various psychological tests were used. Most of the subjects were unusually rugged extroverts of aggressive appearance and temperament. The real psychic troubles in severe Ménière's disease are much more deep-seated than fear of an impending attack. The effect of the psychogenic factors seems to involve slowing and arresting of the small blood vessel circulation within the labyrinth. The vascular flow of patients with Ménière's disease was studied by observing the conjunctival vessels. Clumping of red blood cells and sluggish flow in small vessels were pronounced shortly before and after attacks. Clumping between attacks was more pronounced than in controls. Intravenously administred procaine, which breaks up clumping and speeds circulation in small blood vessels, has altered or stopped tinnitus in several cases. It is suggested that these clumps of blood, when caught in the smaller vessels of the labyrinth, are instrumental

in producing labyrinthine hydrops. Psychic stimuli also induce sludging in patients with Ménière's disease. Patients who broke down dramatically with attacks of vertigo were disturbed persons who had been emotionally unstable for a long time. Psychotherapy is necessary in the management of Ménière's disease, and in early cases psychotherapy may reverse the course of the disease.

Perceptive deafness, tinnitus, and vertigo, separately or combined in the full Ménière's triad, can be due to allergy of the inner ear (Williams [42]). He compares the production of a simple allergic skin wheal with the special conditions found in the labyrinth. The endolymph nourishes the specialized structures within the membranous labyrinth in much the same way as lymph does the ordinary tissue cells. The allergic process may involve the stria vascularis, producing an excess of endolymph high in protein and with an elevated osmotic pressure. Distention of the cochlear duct results. Initially this is reversible with a fluctuating perceptive or even conductive deafness but progresses to permanent cochlear degeneration. Vertigo will result from a similar but separate disturbance of the blood supply of the cristae or maculae of the vestibular labyrinth. There are many difficulties in the diagnosis of Ménière's disease without hydrops, Ménière's disease with hydrops, and cochlear deafness or endolymphatic hydrops without vertigo.

The diagnosis of labyrinthine hydrops can be made on the basis of the loudness balance test and by the discrimination test for phonetically balanced words. When hydrops of any degree is actually present hypoactivity in the involved labyrinth will be found. The treatment has been a modification of Schemm's diet with other measures to help in the elimination of sodium, administration of a vasodilator to decrease arteriolar spasm, and administration of an inhibitor to depress overactive cholinergic nerves.

Richtner [43] studied 53 cases of Ménière's disease with reference to histamine as an etiologic factor and in treatment. Histamine skin tests showed positive reactions according to Atkinson's criteria in about 15% of the cases. The mean blood-histamine level was not higher in vertigo patients than in normal persons and otosclerotic patients; rather, the values were somewhat lower, and lowest in those presenting positive histamine reactions.

Rosen [44] stimulated the intact and sectioned chorda tympani nerve in 36 cases under local anesthesia. Stimulation of the intact or sectioned end of the chorda did not produce any taste sensation. Some dysesthesia was noted. After section of the chorda taste perception disappeared on the anterior two-thirds of the tongue on the same side. In 33 of the 36 cases stimulation of the chorda produced sonic responses variously described as hissing, roaring, buzzing, ringing, etc. In three cases vertigo resulted. Taste could not be evoked by stimulation. The taste system in man includes within it a sonic and equilibratory system very much like the lateral-line organs in fish. This may explain the sonic and equilibratory effects observed on stimulating the chorda.

Surgery in Ménière's disease should be considered only after conservative medical treatment has failed (Frenckner [45]). The ideal operation would be a nondangerous procedure which would relieve vertigo and tinnitus while preserving hearing. He combines fenestration to decrease intralabyrinthine pressure, tamponade of the ampullar portion of the horizontal semicircular canal to destroy vestibular function without injuring the membranous labyrinth, and tympanosympathectomy together with section of the chorda tympani and tendons of the tensor tympani and stapedius muscles to eliminate tinnitus. Fenestration and tympanosympathectomy can be done without disturbing the ossicles.

Surgical destruction of the labyrinth is discussed by Day.[46] Labyrinthectomy is completely effective in terminating the attacks of vertigo in unilateral Ménière's disease. It also relieves distressing cochlear symptoms at the expense of destroying hear-

ing which already is of little or no use. Surgical risks are minimal. Complications are rare and benign. The operation is simple and of short duration. The average patient returns to work within three weeks.

Flett [47] reports his results with the Portman operation in 61 patients with Ménière's disease. Two postoperative deaths were reported (meningitis, bronchopneumonia). Eight immediate failures were followed one week later with destruction of the labyrinth. Perfect results with retained hearing were obtained in 14 cases. Improvement was reported in 36 cases.

Barber and Ireland [48] reviewed 103 cases of labyrinthine hydrops which were treated by differential section of the eighth nerve. Vertigo was relieved in 83%; tinnitus improved in 41.5%. In an appreciable number of cases useful hearing for pure tones was maintained for a limited period; however, half of this group had a significant discrepancy in their reception of recorded speech. They considered this operation very satisfactory for the rehabilitation and relief of the patient with vertigo in Ménière's disease.

Histological changes in Ménière's disease are due to allergic factors, vasomotor disturbances (central or peripheral), infectious foci, or angioneurotic edema (Krejci [49]). When conservative therapy fails or the disease is associated with unilateral high-grade deafness, surgical intervention is indicated. The various procedures (decortication of the common carotid artery; extirpation of the cervical portion of the sympathetic nerve; section of the vertebral artery; labyrinthectomy, or chemical, mechanical, or thermal destruction of the labyrinth) rid the patient of vertigo but at the same time resulted in marked auditory impairment. Krejci demonstrated in animal experiments that ultrasound therapy, diffusely applied to the inner ear, results in high-grade auditory impairment, but when the labyrinth was isolated and treated with a modified applicator (800 kHx: 4 W/cm.^{3}) the function of the vestibular apparatus was eliminated within two minutes, while hearing remained unimpaired. Isolated ultrasound therapy proved superior to electrocoagulation of the labyrinth. It was applied with good success to one patient with Ménière's disease.

Passe [50] reported his results with sympathectomy for various diseases of the labyrinth. A review of the anatomy and physiology of the autonomic nervous system as it affects the labyrinth is given. Gross dilatation of the vessels of the tympanic membrane, walls of the middle ear, and membranous labyrinth follows procaine block of the stellate and upper four or five thoracic ganglia. After stellectomy, block of the thoracic ganglia has no effect on the vessels of the ear. Procaine block of various autonomic nerves and ganglions may influence tinnitus, vertigo, and perceptive deafness. If a good result is obtained by block Passe advises surgical interruption of the sympathetic. If the tinnitus in a case of Ménière's disease is subdued and the low tones improved for air conduction by procaine block, then the case is considered suitable for upper dorsal sympathectomy. Definite conclusions as to the merits of this surgery for nerve deafness have not been determined. Tinnitus which is associated with deafness or vertigo and which is temporarily alleviated by procaine block warrants sympathectomy. More than 80% of the cases were relieved of their attacks of Ménière's disease, and 93% have remained free from major attacks. Tinnitus was more difficult to cure, with one-third showing complete relief.

Hoogland [51] reports results of treatment of Ménière's disease by procaine block of the stellate ganglion. The anterolateral approach of Leriche-Arnulf was used and 20 cc. of 0.5% procaine injected into the area of the stellate ganglion. In severe cases the ganglion was blocked twice a week. In bilateral cases the block was alternated between the sides. Seldom were more than 10 injections needed. Of 37 patients, 60% were permanently improved, 19% temporarily relieved, and 21% did not respond.

Seiferth [52] reports two cases in which marked Ménière's disease was treated with

streptomycin sulfate. Vertigo was completely controlled, but there remained disturbances of equilibrium when walking.

For the purpose of exclusion of the vestibular nerve, streptomycin sulfate was used instead of the more commonly employed dihydrostreptomycin: The latter affects the cochlear nerve earlier than does streptomycin sulfate. Exclusion of the vestibular nerve by means of streptomycin is superior to section of the vestibular nerve, and if periodically checked by audiogram the danger of damage to the cochlear nerve can be excluded. Streptomycin therapy, however, is recommended only for difficult cases of true Ménière's disease which have failed to respond to other medication.

Both sides of the vestibular system are equally sensitive to streptomycin, so if the drug were used in treatment of Ménière's disease vestibular function would be lost bilaterally (Cawthorne[53]). This is easily overcome in children, fairly easily in young adults, and seldom from middle age onward, so that its use is contraindicated in patients over 50 years and in unilateral Ménière's disease. In bilateral cases it should only be considered in young patients when all other measures have failed to control the attacks.

Electrical stimulation of the cervical sympathetic trunk decreased the magnitude of the cochlear microphonic potentials with the use of pure-tone stimuli between 1000 and 2000 cps (Seymour and Tappin[54]). This reduction in magnitude of the potentials is unaccompanied by any change in their sinusoidal wave form. The decrease of the potentials is probably due, in part, to diminution of the cochlear blood supply resulting from vasoconstriction. After cessation of stimulation the potentials mostly regained their original value. The decrease of the potentials is sometimes preceded by a small but appreciable increase. The deafness during attacks of vertigo in Ménière's disease may be due to sympathetic hypertonus.

Lindsay and associates[55] offer the results of experimental destruction of the endolymphatic sac and duct in seven cats, three of which were conditioned by auditory testing. There was no significant abnormality in the cochlear or labyrinthine histology or in the auditory threshold in from three to nine months after the experimental surgery. Successful surgical blocking of the periotic duct in two cats was followed by no histological changes in the inner ears or change in auditory acuity, with time lapses of four to eight months. The authors feel that these experimental results "should not be interpreted to indicate that these structures perform no function," but that "some service may be performed which can be assumed by other structures in their absence." In undertaking the experiment the authors felt that the foregoing conclusions "would detract strongly from those theories which ascribe certain ear diseases (particularly Ménière's disease) to abnormal function of these structures."

Kristensen[56] performed unilateral and bilateral operations on the external semicircular canal in guinea pigs in a manner similar to Cawthorne's operation on the horizontal canal in man for Ménière's disease. Before and after operation hearing (Preyer auricle reflex audiograms) and vestibular function were tested. At first the same phenomena as in cases with total loss of a labyrinth were observed. Later on a rather quick compensation took place, with return to normal of hearing and reactions from the vertical canals. The horizontal canals did not recover. The static function is not influenced.

Acoustic Trauma

Destruction of hair cells in Corti's organ is the primary lesion caused by acoustic trauma (Guild[57]). The outer hair cells are more vulnerable, but a sufficiently large traumatic effect may cause a destruction of inner and outer hair cells alike. Histologically the hair-cell lesion is limited to a relatively short part of Corti's organ. Secondary to the destruction of the hair cells, changes may occur in the ganglion cells of the nerve fibers that terminated on the hair cells and in the nerve fibers themselves. Such changes as do occur are always

limited to the parts of the nerve of hearing that supply the regions of the cochlea in which damage to the organ of Corti has occurred.

Gallagher and Goodwin [58] took 47 subjects with normal binaural hearing and exposed them monaurally to intense white noise (115 db. above threshold) for 10 minutes. Sixteen additional subjects with slight high-tone unilateral hearing losses were given a like exposure in their normal ears. Auditory thresholds for three test tones of 2048, 4096, and 8192 cps were determined immediately before the exposure to noise and at various intervals for approximately 20-30 minutes after exposure. After the exposure, all subjects showed moderate to marked temporary elevation of thresholds at each test frequency, with complete recovery to normal acuity, usually within 30 minutes. Audiograms of the initial postexposure losses showed a decided bimodal distribution at 4096 cps. About one-half of the subjects with normal hearing were hypersusceptible to temporary noise-induced losses, and the remainder were hyposusceptible. The subjects with contralateral high-tone deficiencies fell, with two exceptions, into the hypersusceptible group. The persons hypersusceptible to noise at 4096 cps tended to be hypersusceptible at the other frequencies as well, and rates of recovery could in nearly all cases be correlated with severity of initial loss.

Explosions produce high-tone damage in addition to middle-ear deafness due to perforation of the tympanic membrane, according to Rüedi.[59] The dip at 4096, which is characteristic of noise deafness, is absent in explosion deafness. Immediately after explosion trauma dizziness is noted, with only a gradual recovery. Rüedi exposed guinea pigs to white noise at 100 db. for 1600-2200 hours. After a period of 250 days a hearing loss between 3000 and 10,000 cycles persisted. A localized loss of outer hair cells was observed regularly at the same place, i. e., from the end of the first turn to the middle of the second turn of the cochlea. Guinea pigs were then exposed to the report of a 20 mm. antiaircraft gun at a distance of 2 m. The drum remained intact. At first the pinna reflex was absent for the entire frequency range. After a long rest the responses returned without persistence of any regular loss of function (i. e., C dip). The outer hair cells were destroyed in the same area as that in noise trauma. When pure tones of low frequency are used the histological degenerations, when present, are much less pronounced and they occur more frequently in the upper turns, while high frequencies produce more damage in the first turns. Tones of medium frequency cause areas of severe damage irregularly throughout the cochlea. Some hearing is retained if the first turn of the cochlea remains histologically intact. Pure tones produce much more rapid and widespread hearing losses than noise. Humans were subjected to pure tones at 130-140 db. for three to four minutes. The widespread hearing losses appeared which reached a maximum of one to two octaves above the deafening frequency. Alterations in pitch occurred simultaneously. Pitch is raised by frequencies below 4000 cycles and lowered by frequencies above 4000 cps. Only at 4000 cps is there no alteration in pitch.

Russbach [60] performed difference limen (D. L.) tests in 15 cases of noise deafness and in 13 cases of cranial trauma. Decrease of D. L. was found to be present in new cases of acoustic trauma, whereas D. L. was normal in older cases. In cranial trauma several types of hearing loss may be encountered. Decrease of D. L. was only found occasionally.

Guild [61] surveyed 354 members of the maintenance personnel, particularly those servicing jet engines, to determine if acoustic trauma is a significant problem. The average show some loss over that expected when noise is not a factor. Of those who retain good hearing, most undergo an appreciable shift toward the lower limits of the normal range. Of those mechanics who show significant impairment the degree of severity tends to be greater and the age of onset earlier than is the case among

men who had similar losses in the absence of exposure to excessive noise. Acoustic trauma is likely to become a serious problem with the newer type of aircraft engines.

The diagnosis of early acoustic trauma is not difficult (House[62]). Audiometric tests will reveal a characteristic dip at the 4000 cycle frequency long before the patient becomes aware of any symptoms referable to his ear. The differential diagnosis of more advanced acoustic trauma, after the patient becomes aware of his hearing loss, is much more difficult. Therefore preemployment and routine periodic follow-up audiograms are essential not only to establish an accurate diagnosis but, much more important, to determine the presence of acoustic trauma at the earliest possible moment in order that adequate protective measures may be instituted.

Since ear defenders or wardens offer a means of reducing the incidence of aural casualties, their general use will have to be seriously considered in any future war. Korkis[63] advises the following classification to ensure uniformity in the reporting of blast injuries:

Group I. Lesions of the Middle Ear
- (*a*) Blast contusion
  - Petechial
  - Confluent
- (*b*) Blast rupture
  - Small (one quadrant of drum)
  - Medium (two quadrants)
  - Large (more than two quadrants)
- (*c*) Blast hyperemia (combined lesion)
- (*d*) Blast retraction

Group II. Lesions of the Inner Ear
- (*a*) Cochlea (transitory or permanent)
- (*b*) Labyrinthine (transitory or permanent)
- (*c*) Cochleovestibular (transitory or permanent)

Group III. Mixed Lesions
- Combined lesion involving both the inner and middle ears

Kryter[64] proposes several tentative criteria for industrial noise that is continuous or repetitive. 1. Criterion for voice communications: For perfect work conditions in which person-to-person voice communication at near conversational intensities is required, the noise or "unwanted sound" spectra should not exceed about 40 db. at 2000 cps or for the critical band at that point and even less at higher frequencies. 2. Criterion for deafness: For work situations requiring minimal amounts of voice communication the maximum safe intensity for exposure for indefinite periods is around 85 db. for tones or critical bands in the case of noises of continuous spectra. This 85 db. criterion is an extra-safe figure; further research will probably indicate the necessity for revising this value upward for the frequencies below 1000 cps and above 3500 cps. 3. Criterion for nonauditory work output: Except for possible damage to the ear, regular or expected noise as high as 100-110 db. per critical band can be tolerated with no detrimental psychological or physiological effects, provided the worker has had sufficient time to adapt.

According to Larsen,[65] the degree of hearing loss sustained depends on the following: (1) total duration of exposure, (2) interval between cessation of work and hearing test, (3) quality of noise, (4) vibrations, and (5) the worker's condition (constitutional susceptibility, previous ear diseases, the use of ear defenders, and age). The worker with nerve deafness is more susceptible to noise than one with normal hearing. Tinnitus is present in about 50% of cases. Vertigo is uncommon except occasionally when vibrations are combined with noise. Insomnia (11%), headache (19%), and nervousness (33%) are quoted. Hearing for whispered voice as contrasted to that for the spoken voice disappears proportionately early, so that the vocal index is small (partially due to regression). The upper tone limit is reduced in only 15%. The lower tone limit was elevated in only 10% and perhaps is an expression of bass deafness caused by vibrations. When the differential diagnosis from presbycusis is in doubt the distinction can be based on regression, it being present in all cases of traumatic deafness and absent in presbycusis. General prophylaxis consists of reducing noise at its source (substituting welding for riveting, isolation of noisy machines, sound-absorbing walls, rubber mountings for machinery,

etc.) and organizing measures (rest periods from noise). Individual prophylaxis consists of selection of workers and protection of the ear. A preexisting hearing loss is a contraindication to noise exposure. It is not possible to get an exact picture of auditory fatigue by measuring the effect of fatigue at only one frequency. The response is variable from person to person, from ear to ear, and even in the same ear at different sittings. Also it has been impossible to demonstrate a correlation between the size of the 4096 dip and the size of the auditory fatigue effect.

Theilgaard[66] believes that prophylaxis against occupational deafness may be based on measurements of auditory fatigue. The usual method of measuring auditory fatigue (using the rise of the threshold one-half octave above the frequency of the tone of exposure) is misleading. He emphasizes the importance of inserting an interval between the exposure and the measurement of the effect of fatigue. This avoids the adaptation phenomenon as well as the phenomenon of fatigue in the intermediary and central parts of the sound-perceiving apparatus. He suggests a preemployment test to determine susceptibility to noise. His method is time-consuming, as it requires exposure experiments using four frequencies on each ear. Using noise of known composition instead of pure tones may simplify the determination.

Industries in which loud noises are produced should establish comprehensive hearing programs (Sataloff[67]). The programs should consist of acoustical evaluation of the noise for intensity and frequency characteristics, preemployment hearing studies, and periodic audiograms on all employees exposed to hazardous or potentially hazardous sound. The program should be administered by the industrial physician and the safety engineer.

According to Boynton,[68] methods of noise reduction in noisy industrial operations may be done in one of three ways: (1) use of sound-absorbing materials on walls, ceilings, or panels; (2) enclosure of machines in sound-absorbing housing; (3) elimination of noise in the machine itself from rate of motion of machine parts, the types of gears and drive sprockets, and the nonrotational movement of machine components. The basic instruments required for noise analysis include a sound-level meter, a sound analyzer, and a vibration pick-up.

Willemse[69] exposed 20 normal subjects to 2000 cps of 100 db. for 10 minutes; their threshold at 2000 cps was then determined after 15 seconds, 30 seconds, and subsequently every 30 seconds. A massive dose (300,000 units) of vitamin A was then given orally every day for two to three weeks. After this period the same test was repeated. All subjects showed less fatigue, which manifested itself in a shorter recovery and more definitely in a smaller loss in decibles. This report suggests that vitamin A effectively protects the ear against noise.

Latent hydrops renders an ear vulnerable to noise-induced trauma (Cook[70]). Endolymphatic hydrops in the form of a waterlogged labyrinth often exists in a latent form. The cochlea often shares in extracellular edema, due to excess sodium intake or possibly to individual instability in the control of electrolyte balance. Such a cochlea may cause no symptoms, unless it is exposed to the traumatic influence of loud noise. This condition may explain, at least in part, the phenomenon of noise susceptibility. Questioning of the patient in regard to his salt intake and the nature of sounds which he hears may give a clue to the condition.

Nash[71] reports that the Compensation Commission of the State of New York is considering the following compromise as a means of satisfying claims: (1) No award shall be made until some period (6-18 months) after the claimant is retired or for other reasons permanently leaves the employment of his company. (2) At that designated time the audiometric plateaus obtained will be considered as levels representing permanent hearing losses provided the claimant shows no evidence of simulating or exaggerating his deafness and providing some rectification factor can be used

to appraise the nontraumatic element of deafness resulting from advancing age. (3) Cash payments will be made promptly when the foregoing conditions are fulfilled. This compromise offers the following advantages: (1) The industrial plants will be kept in operation; (2) the worker will have continuous employment and will not be required to remove himself to another locality in an effort to obtain work; (3) deferring payment of awards until retirement will spread an otherwise ruinous expenditure over a period of years and lighten an almost unbearable financial burden on the employer; (4) the worker will get his award on retirement, when he needs it most.

Fox [72] defines noise as unwanted sound, and the type of deafness caused by its action is influenced by many factors, such as (1) intensity and loudness, (2) frequency spectrum, (3) period of exposure, (4) individual susceptibility, (5) age of subject, (6) coexisting ear disease, (7) character of surroundings in which the noise is produced, (8) distance from source, and (9) position of ear with regard to sound waves. Acoustic trauma can be prevented by reducing the noise whenever possible, by rotation of personnel (allowing for more prolonged recovery periods), and by the use of ear plugs or defenders.

Expert testimony must contain (1) the diagnosis of the disease, (2) the etiologic relationship between complaint and occupational hazard, and (3) the degree of impairment of earning capacity (Mittermaier [73]). It must be stated what period of time has elapsed since the complaint developed. Any damage to the vestibular system should be investigated and the degree of compensatory function established. Mittermaier recommends use of a nystagmograph in addition to caloric and similar tests. A roentgenogram is indispensable to detect possible fractures, and bony changes following chronic inflammations. The degree of impairment of earning capacity depends on the binaural ability to hear the spoken word. It is suggested that a high degree of hearing loss or even complete deafness should not be considered as a cause for revoking a driving permit.

Otologic expertise has assumed increasingly greater importance (Bauer [74]). In approximately two-thirds of all cases deafness is due to trauma, while in the remainder it is brought on by diseases causing adhesions, purulent otitis, and occasionally otosclerosis. Patients with marked Ménière's disease or chronic otitis with involvement of the labyrinth are not actually unemployable, but may be unable to find work compatible with their hearing impairment. The problem is more difficult for the city population than for farmers. The solution rests in prophylactic treatment before the patient loses the job he holds. Recommendations include regular otologic examinations of apparently healthy workers, change to a job at which the patient is less exposed to noise, and prescription of a hearing aid.

Symons [75] reports the development of the Compensation Law in New York, with particular reference to occupational deafness. Six medicolegal questions must be answered before any solution to the handling of claims for occupational deafness can be fairly settled. (1) Where are the threshold limits of deafness due to exposure to noise to be drawn? What is a dangerously noisy occupation? (2) Without preemployment audiograms how can cases of nonoccupational hearing losses be recognized as part of an occupational hearing loss? (3) What about hearing losses due to advancing age? (4) What can be done to overcome the obstacle of variable employee acceptance and compliance to the use of ear defenders? (5) If hearing is damaged by occupation and can be restored to normal by a hearing aid should compensation be paid if the employer furnishes such hearing aid? (6) Can a clever man feign or exaggerate loss of hearing when he is given an audiometric examination?

Carter [76] discusses the estimating of percentage loss of hearing in medicolegal and industrial cases. The method should be easily understood, easily and quickly applied, free from complicated mathematical calculations, easily interpreted before any jury,

designed for both ears jointly considered for the whole individual, based on air-conduction data obtained from standardized audiometers, weighted to give preference to the frequency range of the spoken voice, and founded on the best available acoustic and clinical scientific evidence. Carter believes the Fowler-Sabine method (1939) fulfills these requirements. Others found fault with it and the method was revised in 1947. A new revision is being devised which is considering the relative merits of estimating the hearing loss for speech as well as for pure tones.

Toxic Labyrinthitis

Walker[77] reports his findings in cases of tuberculous meningitis treated with streptomycin sulfate (76 cases), dihydrostreptomycin (6 cases), and with a combination of dihydrostreptomycin and streptomycin (the former was given first and then substituted by the latter) (11 cases). Dosage was 50 mg. intrathecally daily and 20 mg. per pound of body weight intramuscularly twice daily. All received aminosalicylic acid. In an initial unselected group of 30 patients treated with streptomycin deafness occurred in less than 37%, of whom 17% were severely deafened. Of the 17 who received dihydrostreptomycin alone or in combination more than 65% were severely deafened. A roaring tinnitus usually appears first, subsides, and deafness gradually ensues. All but 10 of the 93 cases had absent caloric responses, even after 18 months.

Hawkins and Lurie[78] treated cats with 100, 200, or 400 mg. of streptomycin per kilogram of body weight for as long as 60 days. Vestibular function was tested by recording horizontal nystagmus in response to rotation. Cochlear function was tested with cochlear potentials. Streptomycin caused a loss of vestibular function, beginning in 8-10 days. In one of the severely affected cats only a small action potential component was obtained even at very high intensities. However, responses could still be recorded from the auditory area of the cerebral cortex, indicating that the central pathways must be intact. On histological examination the crista of the horizontal canals showed definite degeneration of the hair cells and shrinkage of the epithelium. The utricular macula showed shrinking of the epithelium and scattered degeneration of hair cells. The saccule showed normal neuroepithelium. The organ of Corti showed changes varying from degeneration of internal and external hair cells to complete absorption. The stria vascularis showed atrophy in the severe cases. The spiral ganglion showed degeneration in severe cases. They noted in some sections degeneration of the inner hair cells without a corresponding degeneration of the outer hair cells. This may indicate a special sensitivity of the inner hair cells to streptomycin.

Whetnal and Lucas[79] found extensive tuberculous infiltration of the eighth nerve in the temporal bones of fatal cases of tuberculous meningitis which were treated with streptomycin sulfate and dihydrostreptomycin. They suggest that slight changes in the cochlea could have been caused by disease in the nerve. The incidence of deafness in other forms of meningitis and the rarity of deafness when streptomycin is given for other forms of tuberculosis support the view that deafness may be due to meningitis and not to streptomycin.

Protracted injection of large doses of streptomycin sulfate to guinea pigs produces marked functional damage of the equilibrium apparatus and a loss of sound perception (Rüedi et al.[80]). Changes were observed in the cochlear nucleus but did not seem to be a specific tissue alteration due to streptomycin. The vestibular and cochlear functional losses are mainly correlated with the degeneration in the sensory terminals of the peripheral vestibular apparatus. The severest degeneration of Corti's organ and of the corresponding nervous apparatus as well as the pathological changes of the stria vascularis are found in the lowest cochlear turns. The pinna reflex test and the cochlear microphonic tests appear equally valid for

functional testing. Loss of sound perception was not in proportion to the amount of streptomycin used per kilogram of body weight. The protracted administration of large doses of hydrochloric quinine provokes marked loss of sound perception. This functional damage is histologically correlated with degeneration of Corti's organ, the corresponding nervous apparatus, and stria vascularis. The degenerative processes caused by quinine begin in the external hair cells. The peripheral sensory terminal of the vestibular apparatus remained normal.

Serum neuritis is not a rare complication after administration of heterologous serum (Schwab[81]). It also occurs with antidiphtheric, antiscarlatinal, antianthrax, anti-Pneumococcus, and anti-Streptococcus serum, but is most frequent in antitetanic therapy. In the case of a 59-year-old patient, high doses of antitetanic serum resulted bilaterally in hearing impairment closely approaching complete deafness. Tetanus itself could be excluded as the cause, because the injury involved the lower leg. Serum neuritis is an antigen-antibody reaction affecting specific parts of the nervous system. It is not known why the ear becomes involved, and individual predisposition is assumed. Both ears are equally affected.

Focal infection anywhere in the head but particularly of tonsils and teeth may have a detrimental influence on the level of hearing following fenestration operation (Wullstein[82]). Focal infection has also a detrimental effect on inner-ear deafness traceable to causes other than otosclerosis. Routinely he eliminates all demonstrable foci (particularly teeth) before performing the fenestration operation. With regard to tonsillectomy greater restraint is recommended, because this intervention may result in deterioration of bone conduction, probably due to the administration of adrenergic drugs.

Function and Diseases of the Labyrinth

Severe vertigo is usually due to involvement of some part of the vestibular system (Cawthorne[83]). Central vertigo may be caused by a vascular lesion or sclerosis. Tumors, because of their gradual onset, are not so apt to produce severe vertigo. Vertigo with loss of consciousness suggests an epileptic seizure. A spontaneous nystagmus persisting for more than three weeks is of central origin. Vertical nystagmus is always due to central lesion. Except in acoustic neurinomas, deafness is seldom associated with vertigo due to central causes. Examination of 1523 patients with vertigo showed 107 cases due to involvement of the central vestibular connections and 1349 cases with a lesion of the peripheral vestibular system. Causes of peripheral vertigo include hydrops of the labyrinth; fracture or concussion; infections secondary to otitis media, mumps, or geniculate herpes; labyrinthitis due to toxicity of streptomycin and vestibular neuronitis; paroxysmal positional vertigo. The first step in management of vertigo is to exclude any central cause. Special balancing exercises are invaluable in traumatic cases. During an attack absolute rest and sedation are necessary. Vomiting may require parenteral control (phenobarbital sodium, 3 grains [0.2 gm.]). In Ménière's disease barbiturates and seasickness remedies are useful. Sodium intake should be limited. To influence the blood supply to the labyrinth histamine, nicotinic acid, and operations or injections into the cervical sympathetic ganglions may be tried. When the attacks are not controlled by conservative measures and the disease is unilateral and hearing badly affected, some form of operation on the labyrinth may have to be considered.

Mygind[84] designates an affection in the humoral system of the labyrinth as labyrinthosis. In some patients with Ménière's disease there was histological evidence of increased intralabyrinthine fluid production and solid tissue compression. In severe cases dilation of the endolymphatic system occurs. Although Ménière's disease has been interpreted as a perceptive affection with altered cells in the organ of Corti, a distinct alteration of the nerve fibers or ganglion

cells has never been found. Labyrinthosis may be asymptomatic when bilateral and symmetrical changes neutralize each other, or it may be severe, as in a classic case of Ménière's disease.

Extralabyrinthine phenomena characteristic of vasomotor instability are frequently associated with labyrinthosis. Sedentary habits, poor nutrition, hormonal and psychic influences may be indirectly responsible for changes in the fluid balance. Sudden and permanent loss of hearing with tinnitus and vertigo is probably due to another labyrinthine pathologic process, possibly hemorrhage. Treatment in Ménière's disease is directed against abnormal water metabolism. A low salt diet and restricted fluid intake are prescribed; alcohol and tobacco are banned. Belladonna and barbiturates are helpful in overcoming vertigo. Eustachian catheterization and mobilization of the fenestrae may be beneficial. After convalescence a plain, frugal life with plenty of exercise, fresh air, rest, and a sensible organization of work is essential.

Present-day emphasis in Ménière's disease is placed on distortion of hearing and on the exacerbation of tinnitus and deafness during the attacks (Dix and Hallpike[85]). Loudness recruitment is always present, and adaptation is unusually rapid. A loss of intelligibility at speech tests which is out of proportion to the pure-tone loss may be found in many cases. Two clinical entities are separated from the large group of so-called pseudo-Ménière's syndromes. The first, called vestibular neuronitis, is marked by the absence of cochlear signs, with a vertigo that is not always paroxysmal in character. It affects chiefly the age group of 30-50 years. Often there is evidence of afebrile disease, an infection of the ear, nose, or throat, or only a high sedimentation rate is found. Marked changes in the vestibular tests are present (reduced caloric and galvanic responses). This form of organic vertigo is localized in the vestibular apparatus, central to Scarpa's ganglion, including the nuclei in the brain stem. The condition is benign. The second variety of organic vertigo has positional nystagmus as the chief symptom. The cochlear and vestibular tests are normal. Evidence of ear disease is inconspicuous. No neurological disorders or central lesions were observed. The condition is benign. Symptoms subside with sedatives. The pathology is in the labyrinth, toward which, when undermost, the nystagmus is directed. The lesion affects the otoliths. Disorganization of the sensory cells was found. These chronic tissue changes may be due to trauma, chronic infection, or possibly to vascular disease.

Postural vertigo and positional nystagmus establish the presence of a disturbance of the vestibular apparatus, according to Lindsay.[86] Postural vertigo may indicate a disturbance of either the central or peripheral vestibular apparatus. The positional nystagmus may be direction-changing or direction-fixed. The latter appears only in certain positions but is constant in direction. It may appear only with change of position, or there may be spontaneous nystagmus which is increased in certain positions. Direction-changing positional nystagmus may be maintained or of limited duration. Sometimes the nystagmus appears before the change in position is finished; in other instances it may occur only after the change in position has been maintained for several seconds. Positional nystagmus may be seen in either central or peripheral vestibular disturbances (multiple sclerosis, brain tumor abscess, hemorrhage, embolus, thrombus, inflammatory tubal and middle-ear disease, labyrinthine fistula, and labyrinthitis). A maintained direction-changing positional nystagmus is usually strong evidence of a central vestibular affection. Direction-changing nystagmus of limited duration may be found in the type of inner-ear disease in which there are sudden onsets of high-tone hearing loss and vertigo in a previously healthy ear. Positional nystagmus is not often present in Ménière's disease but is in pseudo-Ménière's syndrome (in which the classic auditory phenomena are absent). The commonly seen cases of positional nystagmus are difficult to localize centrally (intoxica-

tions, infections, hypertension and hypotension, menopause, vasomotor instability, and head trauma). The caloric response is normal. Therapy is directed at the causative process. Symptomatic relief may be obtained with barbiturates and drugs which appear to alleviate motion sickness.

Wodak [87] uses Demetriades-Meyer's method for stimulating the labyrinth. Latent periods shorter than 10 and longer than 30 seconds are considered pathological. Peripheral hyperexcitability is diagnosed when the latency period is shortened to 10 seconds or less, the tonic reflexes (nystagmus, deviation reactions, arm tonus reaction) being as a rule very strong. In central hyperexcitability there is a normal latency period (15-20 seconds) and prolonged (3-5 seconds) and exaggerated tonic reflexes. Peripheral hypoexcitability is diagnosed when the latent period lasts much longer than normal (30 seconds or more) and the tonic reflexes are very weak or shorter than normal. Central hypoexcitability has so far been almost impossible to diagnose.

The Vilstrups [88] measured the postural changes of the utricular otoliths with x-rays of decapitated sharks. All photographs were completed within five minutes of decapitation. A metal wire inserted along the medial wall of the cranium served as a fixed measuring point. Upon rotating along a transverse axis definite changes in position were found. The otolithic membrane does not quite follow the rotation of the cranium but lags behind, i. e., trying to remain in the same place when the cranium moves. This movement, which amounts to about 2 degrees, takes place in the subotolithic clear zone between the otolithic mass and the border of the sensory cells. A corresponding tendency to remain in situ was found on postural changes after rotation round a longitudinal axis. The studies confirm the idea that a shift in the effect of gravitation upon the otolithic membrane brings about a deflection of the projecting parts of the sensory cells (cilia), resulting in the transmission of nerve impulses.

Jongkees [89] stresses that too strong a stimulus should be avoided in performing the caloric test. At 30 C and 44 C the vestibular reactions are nearly maximal and the well-being of the patient is not disturbed. Duration is the only quality of caloric nystagmus giving a reliable index of its magnitude. The duration of caloric nystagmus is not of pure vestibular origin. Fixation of the eyes may influence nystagmus greatly and should be eliminated with Frenzel's glasses. The double bilateral caloric test (cold and warm water in both ears) can differentiate between a predominance of the function of one labyrinth and a directional preponderance of the nystagmus. Predominance of one labyrinth occurs in 5% of normal subjects; directional preponderance occurs in 17%. Bárány's test is of limited value because of too vigorous stimulation. Veit's modification uses a subliminal acceleration; when a constant rotational velocity is reached the chair is stopped and one pure stimulus, the deceleration, acts on the vestibular system. If one pure stimulus is given, the sensation of turning is very distinct and may be measured. There are two measurable responses to vestibular stimulation, nystagmus and sensation. The durations of these two phenomena plotted for various stimuli of different intensities on a logarithmic scale give an easily read graph, the cupulogram.

Lowenstein [90] recorded the responses from individual end-organs of the vestibular apparatus by using oscillographic recording of action potentials from sensory nerves. Recording from the branch of the eighth nerve supplying the ampulla of the horizontal canal while the animal was rotated proved that the canal response to angular acceleration is inherently bidirectional. This response is based on the presence in the resting animal of a spontaneous discharge of action potentials, the frequency of which in the horizontal canal can be increased by ipsilateral and decreased by contralateral acceleration. The tonic function of the semicircular canals was demonstrated in pike by severing the nerve supply to the horizontal canal and

showing in the surviving animal the existence of well-circumscribed, lasting tonus asymmetries of the eye-muscle apparatus. Single fiber studies led to a quantitative description of the behavior of the cupula terminalis and proved the torsion-pendulum theory of cupular function. All three otolith organs—utricle, saccule, and lagena—participate in maintenance of equilibrium. Vibration responses were recorded from nerve fibers leading from parts of the macula sacculi and neglecta and the lacinia of the macula utriculi. The otolith-bearing part of the macula utriculi, the posterior portion of the macula sacculi, and the adjoining macula lagenae do not respond to vibrations.

Boenninghaus[91] reports on the effect of dimenhydrinate (Dramamine) on experimentally produced nystagmus and vertigo. Both nystagmus and vertigo arise from the vestibule but are not necessarily related; furthermore, the vestibular apparatus is subject to the influence of higher centers as well as of the vegetative nervous system. For this reason drugs may act selectively on the two conditions. In serial experiments it was demonstrated that dimenhydrinate diminishes nystagmus (more precisely its postrotatory forms I and II) with regard to duration as well as to intensity of subjective feeling. This finding is in contrast to the effect of small doses of phenobarbital (Luminal) and hexobarbital (Evipal), caffeine, Vasano (a scopolamine preparation), etc., all of which diminish vestibular action without at the same time mitigating experimentally produced nystagmus. Boenninghaus explains the effect of dimenhydrinate as due to inhibition of cerebral centers of vestibular function. This assumption is confirmed by the finding that the drug also inhibits the center of emesis and other vegetative centers, and causes a feeling of tiredness.

Vertigo refers to all sensations of disequilibrium arising from vestibular disease (Symonds[92]). Three varieties exist: paroxysmal, chronic, and postural. Lesions of the vestibular nuclei have a persistent and often progressive chronic vertigo with nystagmus. Vestibular neuronitis is characterized by single or recurrent attacks of paroxysmal vertigo without auditory symptoms. Vertigo as an epileptic aura is not present after return of consciousness.

Lewis[93] reviews the anatomy and physiology of the sympathetic nervous system outside the central nervous system. The influence of sympathectomy upon the function of the internal ear is discussed. The segmental sympathetic supply to the internal ear is unknown and the sympathetic pathways in the neck have not been fully mapped out, so that one can never be certain of completely interrupting its sympathetic supply. Stellate ganglionectomy is more likely to succeed than either Telford's or Smithwick's operation because the supply from T 1 is interrupted as well. This operation is predominantly a preganglionic section of sympathetic nerve supply to the cephalic region. Other possible pathways are left intact, and if improvement occurs after cervicothoracic ganglionectomy and then relapses, it would be worth considering the removal of the superior cervical ganglion and a length of the cervical chain in order to interrupt any residual fibers. A preliminary injection of procaine would help in deciding whether a successful result could be expected. If it is shown that the internal ear receives sympathetic fibers from both sides of the body through anastomosis on the basilar artery and the circle of Willis, a decision will have to be made as to whether or not bilateral operation is justifiable.

Any good results that follow operation appear to be due to the prevention of vascular spasm rather than to the production of vasodilatation. Although immediate relief of vertigo in Ménière's disease has resulted from different forms of sympathectomy, it may prove impossible to achieve lasting results because of the return of vascular tone, which occurs even after complete sympathectomy. It is probable that sympathectomy affects the labyrinth through its vascular supply and when vascular tone reappears the unknown pathological factors

which cause symptoms may become effective again.

Lauter[94] states that dizziness is an ill-defined complaint. While psychosomatic factors cannot be excluded, the diagnostic investigation is initially concentrated on organic causes. As a first step, ophthalmologic causes must be eliminated, particularly errors of refraction and disturbances of the ocular muscles. Impissated cerumen may cause hearing loss and vertigo. In the absence of such findings we have to think of catarrh or occlusion of the Eustachian tube. The most frequent and difficult type of dizziness is related to the labyrinth and the vestibular system; associated nystagmus is a characteristic sign. Dizziness may also occur in otosclerosis. It is one of the symptoms of intracranial disease and is particularly observed with cerebral injury, multiple sclerosis, and certain types of tumor. Vertigo associated with tabes may simulate cerebral involvement. Dizziness is often due to circulatory disturbances (anemia, sclerosis, hypertension, Ménière's disease, migraine, poisons). Other causes are petit mal, and neurotic states.

Hearing Tests and Auditory Training

Hirsh[95] correlates audiology and those scientific areas to which audiology is most intimately connected. The several clinical activities in the field of audiology are (1) measurement of hearing; (2) diagnosis; (3) prognosis for surgical, pharmaceutic, physical, or educational therapy, and (4) therapy itself. A hearing aid by itself can rarely provide as much benefit as it can when it is used in cooperation with an orienting program of auditory training and lipreading. Clinical audiometry in the past has been characterized by only one kind of measurement, namely, the absolute threshold. Additional measurements must be made for prognosis with regard to a hearing aid, surgery, or particular educational procedures. These include speech audiometry and the examination of such psychophysical functions as loudness, masking, and fatigue. Prognosis for lipreading is still a difficult problem. Auditory training is achieving an importance that is relatively new. Audiology must look further to psychiatry and clinical psychology for another kind of prognosis and diagnosis. The loss of hearing has ramifications that go beyond the loss of communicative skill to basic changes in the personality.

Metz[96] employing an acoustic impedance bridge observed the contraction of the middle-ear muscle in various types of hearing loss. In patients with impaired hearing of the peripheral perceptive type the threshold value for eliciting contraction of intra-aural muscles is the same as that of persons with normal hearing; thus complete recruitment of loudness occurs. If there is a perceptive deafness due to a lesion of the nerve (acoustic neurinoma) the reflex contraction of the muscle does not occur, nor does it occur in otosclerosis. Both the recruitment of loudness and the preserved threshold for eliciting the muscle reflex are attributed to the same cause (i. e., the same pathological changes in the organ of Corti).

Tiffany and Hanley[97] studied the effect of delayed feedback of their own voices in 30 speech pathology students with normal hearing. Despite efforts of the subjects to overcome the effect of delayed speech, significant retardation in reading rate resulted which increased with increasing intensity of the delayed side-tone. The use of the delayed feedback speech phenomena is feasible in the detection of malingering and/or psychogenic deafness.

Kristensen and Jepsen[98] discuss recruitment in otoneurologic diagnosis. In Ménière's disease recruitment is a constant finding in the defective ear if its hearing is reduced. In acoustic neurinoma if sufficient hearing is retained in the defective ear to allow recruitment determination, it was found that recruitment was absent. Perception deafness without recruitment is of very important diagnostic significance, since it makes possible an earlier diagnosis of acoustic neurinoma, which accounts for about 10% of all intracranial tumors. Perception deafness without recruitment has

also been reported in cases of cochlear neuritis and head trauma. Absence of recruitment indicates a retrolabyrinthine disease.

Jerger[99] presents the Northwestern difference limen (DL) test for recruitment, which is performed at a sensation level of 15 db. using an ascending method of stimulus presentation without beat interruption. Degree of modulation in the beat could be varied continuously from 0 to 5 db. and was accurate to 0.1 db. The test was standardized on 39 normal ears. Compared with that of the normal hearing at equivalent sensation levels the DL was normal in pure conductive hearing loss, indirectly supporting the hypothesis that recruitment phenomenon and DL size are related. A group of 74 cases of sensorineural loss with or without a conductive component were tested. The DL's were abnormally small in cases of end-organ damage (acoustic trauma and endolymphatic hydrops). DL's were normal in the retrolabyrinthine lesions of the cerebellopontine angle.

Huizing and Reyntjes[100] studied recruitment and speech discrimination loss. Two articulation curves have been previously noted: the C type, indicating pure sensitivity loss (conductive) in which hearing may be restored to normal by adequate amplification, and the P type, combining sensitivity loss with discrimination loss due to loss of perceptive hearing. To these, the authors add a third, the R type, which combines sensitivity loss with loss of discrimination by subjective distortion due to recruitment. The R type curve is distinguished from the P type curve because of the predominating influence which speech intensity exerts on articulation score. This is specific for recruitment. P type hearing has a constant discrimination loss, whereas the R type has a variable loss. When the optimal listening level (80 db.) is passed the articulation score rapidly falls. Alteration of the shape of the R curve with a hearing aid depends on speech intensity and on the type of frequency response characteristically used. When subjective distortion originates from a narrow band of the tonal scale, suppression of the band may change the R type curve to a P type in which saturation level exceeds optimal level of the original. Frequency response characteristically should be chosen so that the center of intelligibility is moved to a part of the frequency level in which subjective distortion is as low as possible while the contributing band width remains large enough to obtain a better condition.

Ascertainment of the need for special educational treatment involves very much more than a measurement of hearing loss (Watson[101]). It necessitates a diagnosis of the child's existing condition and a prognosis of the type of education which will be best suited to his handicap. Children with impaired hearing should be tested on the threefold basis of hearing loss, educational attainments, and intelligence in order that the appropriate special educational treatment may be given.

In another paper Watson[102] states his experience with the fact that 95% of pupils in schools for the deaf have some residual capacity to hear and about 70% have useful residual hearing. He advises in all cases to test whether or not a hearing aid will be of any value. The essential feature of auditory training is the combination of aided hearing and lipreading so as to reach the child's mind by as many channels as possible. Group aids are more useful in work with severely deafened children, but with the less severely deaf child aids are probably more useful because they can be used outside the classroom as well. The optimum time for the development of speech and language in deaf children is the same as in hearing children. For children with only partial deafness it is absolutely essential to their education to receive as much auditory training as can possibly be given, and for children classified as deaf and dumb training is of value in about 70%.

Hardy, Pauls, and Bordley[103] state that three basic concepts of rehabilitation are to be considered. First, the child must be considered as a whole; loss of hearing is a disturbance affecting the whole behavior complex. Treatment includes auditory train-

ing, speechreading (lipreading), and speech training with behavioral guidance. The second concept is in the emphasis given to a multilateral approach to this problem (otologist, educator, neurologist, psychiatrist, clinical psychologist, and audiologist). Third is the concept that training and treatment should be started as early as possible in a child's life.

Hearing impairment usually results in unfavorable changes in behavior patterns, which in turn is manifested by serious social displacement of the person (Bergman [104]). Adequate rehabilitation requires the knowledge and skill of many professional workers operating as a rehabilitation team. Bringing this combination of special services to bear successfully requires careful planning by the audiologists, determination and the will to succeed on the part of the patient, a developing community awareness of the problems and needs of persons with impaired hearing, and, finally, financial support.

In another report Bergman [105] has noted that without organized auditory training many persons who try to use an aid are easily discouraged and discard their instruments, while those who finally do make their adjustments take a long time before they become good users. A program of auditory training deals with the following specific problems: (1) developing discrimination of speech in noise, (2) developing efficient listening habits, (3) developing an understanding of the hearing aid as an instrument, (4) developing discrimination of speech sounds, and (5) developing tolerance for loud sounds. Auditory training, speech correction, speech reading (lipreading), and other methods should be offered in a highly organized and concentrated full-time course.

Dix and Hallpike [106] report the advantages of their peep-show technique: (1) Pure-tone audiograms facilitate making recommendations for special educational treatment. They are obtainable by the peep-show procedure in nearly all subjects of normal intelligence in the age group 3-6 years. (2) The test is carried out rapidly, seldom exceeding a half hour. (3) The technique is simple and does not require long or difficult training of examiners. (4) Stress is laid on the well-known practical advantage of the pure-tone audiogram for subsequent check on the progress of the deafness. (5) The test is a good guide of the subject's mental capacity. Children with a loss up to 30 db. do not need special handling; those with 30-40 db. loss may remain in the regular school but require assistance in the form of special seating, lipreading, or hearing aids. Those with 40-80 db. loss require special schools for the partially deaf. With 80-100 db. loss they require special schooling and hearing aids may be of value. Losses of over 100 db. require special schooling for the totally deaf; hearing aids are of no value.

Hall [107] states that parents should be instructed to report otological disturbances in their children to their family physician as soon as possible. The medical students should be grounded in those things which are to form the commonest part of the doctor's daily work. Whenever possible deaf children should be fitted with a hearing aid, which greatly benefits their personality, speech, and well-being. Deafness is somewhat related to the speed of recovery from otitis media. Children are better treated, from the outlook of the final hearing result, by efficient sulfonamide treatment than by poor and intermittent penicillin treatment. Audiometric studies were made in 115 cases of acute otitis media for two years. About 40% were treated with sulfonamide, the remainder with penicillin. In the majority of cases satisfactory audiograms were obtained in two to three weeks. The loss of hearing was greater on the average in the high tones and was recovered first in the low tones. Only 5% had persistent hearing loss two years later. Persistence of the otorhea into the subacute stage demands an urgent priority for tonsillectomy and adenoidectomy. The various methods of operative procedure used in the treatment of chronic cases are discussed. Using the "extended Schwartze operation" he obtained dry ears in 89%, improved the hearing in

76.9%, maintained it in 15.3%, and diminished it in 7.8%. Using the modified radical operation he obtained dry ears in 64.6%, maintained hearing in 20.0%, and decreased it in 13.4%.

If hearing loss is less than 30 db. in one or both ears, it is necessary to make a careful appraisal of the patient and his hearing problem before a decision in regard to the use of a hearing aid can be made (Carson [108]). The disability of unilateral deafness manifests itself by the greater susceptibility of the single ear to the masking effect of noise. This makes for confusion in group conversation. A loud tinnitus frequently occur in the affected ear which may be so intense as to introduce a certain masking effect upon the better ear. Neither the average hearing-aid users nor the patient with a unilateral hearing loss has the ability to localize sounds. Even if there is enough residual hearing in the deafened ear to make possible the use of a hearing aid, it is impossible to balance a normal hearing ear and an aided ear. Patients with a slight hearing loss are under a greater nervous strain than those with severer losses, for many have not yet accepted their handicap and are still making a real effort to hear. Many of these patients with slight hearing loss have extremely annoying tinnitus. Often the use of a hearing aid reduces or eliminates this tinnitus both for these patients and for the patients with severer hearing losses. Because of the psychological aspects involved it is impossible to make the determination as to the recommendation of a hearing aid for these patients by means of standard tests. Each must try a suitable aid (moderate gain instrument). The age, desires, and nervous temperament of the users are the important factors.

Miscellaneous Contributions on Deafness

Gerrard [109] acknowledged the roles of maternal rubella and heredity in the production of congenital deafness. Some cases of perceptive deafness (confused with the congenital type) are acquired soon after birth. In these the deafness is associated with lesions of the cochlear nuclei and can be distinguished by its frequent association with a severe neonatal jaundice, Rh incompatibility, or prematurity. Of 407 children in a school for the deaf, this type of deafness was found in 5 children with kernicterus due to Rh isoimmunization. In 17 additional cases there was a comparable perceptive deafness associated not with Rh isoimmunization but with a high incidence of severe neonatal jaundice and prematurity.

Intracranial injuries to the newborn resulting from cerebral anoxia and cerebral hemorrhage may give rise to permanent neurological symptoms (Johnsen [110]). Perceptive deafness is shown to occur more frequently in the first born than in children with older sibs. Perceptive deafness is more frequent among children born prematurely than among children born at term. Of 39 children with perceptive deafness 6 had shown symptoms of asphyxia immediately after birth. A strikingly high number of children born at term and with subsequent perceptive deafness had been delivered in such a way as to imply an increased risk of cerebral anoxia and hemorrhage.

Johnsen and Freiesleben [111] give a survey of the symptoms and occurrence of kernicterus in cases of erythroblastosis, together with a review of previous reports concerning the connection between perceptive deafness and erythroblastosis. They present 49 children with perceptive deafness showing a statistically significant preponderance of Rh-positive children with Rh-negative mothers, 10 of these children presenting the blood group constellation characteristic of erythroblastosis. Among these 10 mothers 5 showed definite evidence of Rh immunization. The perception deafness is most likely due to Rh immunization and may remain as the only permanent evidence of previous erythroblastosis.

Australian records show that deaf-births have been more numerous in some years than in others and that in the year preceding these years of high incidence epidemics of

rubella have been remarkably prevalent (Lancaster [112]).

The syndrome of embryopathia rubeolosa, characterized by congenital defects of the ears, heart, lens, and teeth, is caused by rubella virus contracted by the mother during the first trimester of pregnancy (Nager [113]). The characteristic lesions are located in the inner ear and consist mainly of collapse of the cochlear duct and of anomalies of Corti's membrane and stria vascularis. The ganglion cells and nerve fibers of the cochlea and vestibule remain intact. The rubella virus affects special tissues in their sensitive phase of development and so is called an infectious disease of the embryo.

The first symptom of senile deafness is tinnitus (Novotny [114]). Otoscopy demonstrates no abnormalities, but auditory tests show that the range for whispering is relatively more restricted than that for normal conversation. At about age 50 it is physiologic that perception of high frequencies decreases, but the loss is noted only when lower frequencies also become involved. The pathologic-anatomic basis rests in degenerative processes in the ganglia of the cochlear nerve. Subsequently there occurs arteriosclerosis in the arterioles of the cochlea, resulting in degeneration of the neuroepithelium of the organ of Corti. For the nervous type of presbycusis, vitamin therapy is indicated. Hearing aids do not improve the situation in the presence of extensive loss in high frequencies, but a trial with air conduction may be attempted; bone conduction always remains unsatisfactory.

Kobrak [115] finds the cause of presbycusis in hypofunction of the muscles of the inner ear. Lack of tonus in these muscles results in the paradoxical finding that sound of above average intensity is insufficiently dulled, and therefore irritating; on the other hand, sound of average intensity is not clearly discernible. Auditory hyperesthesia and hypoacusia thus exist side by side. Similarly the easy fatigability of old people is due to decrease in the tonus of ear muscles, and should not be explained as purely psychosomatic. The clinical syndrome of presbycusis is in all probability different from nerve deafness of young people, traceable to cochlear degeneration. The differential diagnosis is based on the "physiologically negative Rinne test." Presbycusis is regarded as a distinct clinical form of "schizacusis," i. e., disproportion between the hearing of pure tones and of mixed tones (spoken words). Diminished muscular tonus is ascribed to neurovegetative processes, but the histomorphological aspect remains to be clarified.

Senile atrophy of the spiral ganglion and angiosclerotic degeneration of the inner ear are two diseases of the auditory apparatus peculiar to old age (Saxen [116]). Senile atrophy of the spiral ganglion is a process of wear affecting the proximal parts of the cochlea. The organ of Corti is not involved in the process, nor are there changes in the inner-ear vessels. Angiosclerotic degeneration is the result of sclerosis in the corresponding blood vessels. Regressive changes then appear in Corti's organ. In more advanced cases the supporting tissue is also involved. Presbycusis may also be due to lesions in the pathways of hearing and acoustic centers developing in old people, probably on an arteriosclerotic basis. In most of these there is an associated senile atrophy of the cochlear ganglion.

Johnsen [117] studied 80 children with endogenous perceptive deafness and their relatives. He distinguished two groups: (1) a large group comprising mild and severe cases of the nature of defects, not progressing and transmitted by recessive inheritance, and (2) a smaller group made up of mild and severe progressive cases transmitted by mendelian dominance.

Meyer zum Gottesberge [118] states that in the majority of cases nerve deafness is due to degenerative processes affecting the nerve components of the inner ear. The functional character of inner-ear deafness is demonstrated by two facts: (1) Auditory loss increases with higher frequencies and (2) the phenomenon of recruitment, i. e., the

reduction of auditory loss, is observed with mounting intensity. The recruitment phenomenon is a sign of inner-ear disease and occurs only exceptionally in diseases of the nerve stem or of the central auditory pathways. It is possible that hydrodynamic changes in the cochlea are responsible for abnormal strain on the auditory organ.

Bordley and Hardy [119] studied the etiology of deafness in 296 children. In 104 cases the cause was not determined. There were 22 cases of prenatal rubella, 12 of Rh incompatibility, 15 of rubeola, 1 of mumps, 14 with a family history of deafness, 2 due to toxemia of pregnancy, 12 to congenital malformation, 24 to birth injuries, 15 with cerebral degeneration, 5 with severe cyanosis after birth, 14 with unexplained hyperpyrexia, 27 due to meningitis and encephalitis, 2 postnatal jaundice, 2 infant diarrhea, 1 anemia, 3 pertussis, 3 varicella, 1 scarlatina, 1 typhoid, 1 plumbism, 1 citronella poisoning, 12 middle-ear disease, and 2 immunization during pregnancy (measles). Adequate preventative medical measures could have avoided more than half of the above cases of deafness.

Perlman and Leek [120] studied 11 cases of late congenital syphilis of the ear. It affects young women particularly and is often accelerated by pregnancy. Serology was positive in seven cases. Interstitial keratitis had occurred in 10 cases, Hutchinson's teeth in 4 cases. Age of onset was 9-35.

Auditory and vestibular symptoms and results of functional tests are compatible with bone and labyrinth pathology. Main features of the disease are bone lesions of gummatous osteomyelitis. They may invade the labyrinth to produce serous labyrinthitis, with miliary gumma in the endosteum, dilation of the cochlear duct, and, finally, pronounced degeneration of the cochlear and vestibular end-organs. A positive fistula test with an intact normal drum is a distinctive sign. Response is more readily obtained with negative pressure in the external canal, producing a characteristically brief nystagmus. The disease is resistant to antiluetic therapy and may even progress rapidly to profound deafness during intensive treatment. Treatment with penicillin and corticotropin, when given in early stages, appears to offer some prospect for improvement.

Phillips [121] reviews the otologic symptoms in multiple sclerosis which commonly involve the vestibulocerebellorubral pathways. Initial symptoms such as tinnitus or vertigo may subside for as long as five years. In 60% the disease is progressive from the start. Vertigo was present at the onset in 35%, headache in 25%, dysphagia in 20%, and tinnitus in 15%. Occasionally severe nerve deafness is observed at the onset of the disease (auditory nerve or cortex in auditory radiation involved). The commonest symptoms are double vision and weakness or paralysis of an extremity. Careful neurological evaluation will determine whether or not a dysfunction is due to a single or multiple defect. Phillips stresses early diagnosis so that vasodilator drugs (histamine) can be instituted. Antihistaminic drugs are contraindicated.

Asher [122] divided 63 cases of athetosis into three groups: (1) 22 cases with a history of icterus gravis neonatorum (erythroblastosis fetalis), (2) 12 cases following neonatal jaundice without evidence of isoimmunization, and (3) 29 cases without history of jaundice. Hearing defects were frequent in the first two groups and uncommon in the last. This suggests that jaundice results in a common area of brain injury whether due to isoimmunization or not. It is important to be aware of the frequency of deafness in athetosis so that intelligence may be fairly evaluated.

Brown, Love, and Adams [123] reviewed 150 cases of acoustic neurofibroma. This is the commonest lesion in the cerebellopontile angle and constitutes 7%-10% of all brain tumors. Some degree of deafness was present in all cases. Usually it was complete or severe, but occasionally it was mild. In cases with mild deafness labyrinthine function was found to be more severely depressed than cochlear function. A nonfunctioning vestibular labyrinth on the side of the neurofibroma with retained activity of the vertical

and horizontal semicircular canals of the opposite side was the usual finding on caloric stimulation. Deafness, tinnitus, or both were initial symptoms in 70%. The hearing loss progresses rapidly. Because of the slow growth of the tumor and compensation, vertigo was not common. Only 35% complained of some type of dizziness. Spontaneous nystagmus was present in 83%. It is usually not present when the patient looks straight forward but rather on lateral gaze. Corneal anesthesia was present in 84%, and it was bilateral in 12%. Headaches were prominent and progressive and located in the ipsilateral occipital region. Facial paresthesia occurred in 51%, facial weakness in 37%, Facial pain in 11%, and choked disc in 49%. Roentgenograms were helpful in 43%. Disturbed cranial nerve function on the contralateral side is due to the great torsion of the brain stem.

Engström and Graf [124] recorded ear murmurs with calibrated phonocardiography in 12 subjects. Causes of objective tinnitus of a vascular type include arterial aneurysms, anomalies of the vessels on the venous side, stenosis of the carotid or other cranial arteries, arteriosclerosis in the basilar artery, murmurs derived from the heart, inflammatory hyperemia of the ear or its surroundings, and essential objective tinnitus without proved cause.

Baron [125] studied the effect of parenteral vitamin A in 36 cases. The response of deafness (8.3%) or of tinnitus (23.5%) to the drug was very low and not superior to that of other forms of therapy. Baron believes part of the improvement obtained is due to increased resistance to fatigue and to psychogenic factors.

Lawrence and Wever [126] exposed four cats to a mixture of nitrogen and air (oxygen content 0.5%). The magnitude of stimulus required to produce 1μ of response at 12 different frequencies was estimated before and after exposure for 30 minutes so as to determine the differential change in sensitivity. The sensitivity curves show that deterioration of cochlear potentials due to anoxia is independent of frequency. Histologically the basal end of the cochlea showed more changes than the apical end. Various levels of Corti's organ showed greater degeneration than contiguous areas immediately above and below. The stria vascularis varied from normal to completely degenerated and appeared to have no relation to the condition of the adjacent organ of Corti. In Corti's organ supporting cells succumbed before hair cells. The Deiters cells are most vulnerable, the Hensen and Claudius cells less so. The functional efficiency of the hair cells appeared to be well maintained until the later stages of the degenerative process. Final impairment to cochlear potentials was to the extent of 13 db., or a loss of 78%. Another animal was killed with curare, and three hours later the loss in cochlear potentials was 16 db. It appears that Corti's organ retains a good deal of its viability even three hours after cessation of the heart and would make a substantial recovery of function if oxygen were again provided. A more violent death brought about by clamping the trachea produced a loss in potentials of 34 db. three hours after the heart stopped. This greater loss was probably due to a more rapid consumption of oxygen from the greater muscular activity in this form of death. Apparently, nonpathological changes in Corti's organ depend not only on the length of time elapsing between death and fixation but also on the nature of the death and the physiological state of the animal at the time of death.

Portmann et al.[127] studied the effect of supersonic waves on the auditory organs. A series of guinea pigs, rabbits, and rats were treated for 10 minutes with supersonic waves of a frequency of 1,000,000 cps and intensities of 0.5-10 w/sq. cm. The waves were applied through an ear speculum filled with water and placed in the external auditory canal. In the guinea pig the functional effect was obtained by Preyer's reflex. Intensities of 0.5-1 w/sq. cm. were ineffective. An intensity of 2 w/sq. cm. was associated with marked impairment of the function of the exposed and, to a smaller degree, of the unexposed ear. At 3 w/sq. cm.

this was even more marked. Low intensities produced hyperemia; higher intensities gave rise to extravasation. Cellular destruction in Corti's organ was observed at an intensity of 8 w/sq. cm. The supporting cells were those mainly affected. Degenerative changes in the nerve tissues of the labyrinth were produced by 3 w/sq. cm. Since supersonic waves spread as cylindrical bundles in relation to the plane of application, the changes were restricted to relatively small regions, varying with the direction of the bundle. Interference phenoma give rise to so-called mute zones in the course of a bundle of waves; the biological effect is at a minimum within these zones. The most marked changes are observed at the planes separating tissues of varying density.

Schuknecht and Neff [128] produced limited damage to structures within the apical coil of the cochlea in cats. Audiograms were recorded before and after the lesions were made by using the conditioned responses. The results obtained indicate that, at near threshold intensities, frequencies below 500 cps excite receptors only in the apical turn of the cochlea—at least it is only in this region that the effect they produce is sufficient to set off the neural activity necessary for sensory discrimination. As intensity is increased the low frequencies appear to arouse activity over a wide area of the cochlea, so that at intensities 20-30 db. above threshold they are producing excitation at least as far toward the base as the lower middle turn of the cochlea.

Zöllner [129] reports a technique of plastic correction of the labyrinthine windows. After radical operation of the middle ear or in the presence of extensive inflammatory scars it is difficult to obtain a satisfactory degree of hearing improvement by means of a fenestration operation. For the purpose of a specific diagnosis as well as to create a window the recesses are carefully exposed. For dissection in the neighborhood of the stapes Zöllner uses a scissors-like instrument of his own design. When the layers of granulation or scar tissue are removed it is often possible to detect an intact stapes which can be mobilized and will then again function satisfactorily. Sometimes it is necessary to place a prosthesis upon the oval window. In performing this plastic operation one should avoid development of a tissue layer involving both windows. A great advantage of this plastic operation is that the cavity created by radical operation of the middle ear is rendered completely dry.

Shambaugh [130] reviews the embryological development of both the sensory end-organ in the inner ear from the ectodermal otocyst and the sound-conducting apparatus in the outer and middle ears from the gill structures. The anomalies of the outer and middle ears fortunately tend to be independent of anomalies of the inner ear, since they come from different embryonic enlages. The embryo is vulnerable to developmental anomalies of the sound-conducting apparatus at any time within the relatively long period during the second to the seventh month of fetal life, and the particular deformity occurring in the individual case depends upon the time of arrested development and the area of the gill structures chiefly involved. The author feels that variable anomalies preclude a single technique for surgical correction, and that the surgical procedure for constructing the most nearly normal functioning sound-conducting apparatus depends upon the degree and combination of developmental malformations. Thus, a partial canalization of the external auditory meatus with an intact and normal tympanic membrane and ossicular chain is corrected by establishing a patent bony meatus lined with skin grafts, whereas a complete absence of any vestige of an external canal with a bony plate instead of a tympanic membrane and a fused and malformed incus and malleus necessitates a much more extensive creation of a meatus and, after removal of the bony tympanic plate and fused incus and malleus, the placement of a skin graft against a mobile stapes. Fenestration is considered as a primary procedure only in cases of congenital stapes fixation which may be suspected and are confirmed at surgery. Of 22 cases operated on by

Shambaugh and his associates using the variable surgical procedures, 11 have had maintained improvements at the practical 30 db. level or better.

Pattee [131] advocates surgery in all bilateral cases of congenital atresia of the auditory canal in an attempt to improve hearing. A permanent improvement in hearing can be expected in 70% of cases. Only half of these are brought up to an average loss of 30 db. or better. The first operation should be done in such a manner that a secondary fenestration operation can be done in those cases where the hearing fails to reach the serviceable level. Secondary atresia may be obviated by removing as much bone as possible and using a large skin graft. A survey of 64 cases operated on by 31 otologists is reported.

Moritz [132] attempts through a plastic operation in the middle ear to reestablish a protection of the round window against air-borne sound waves. This approach is based on the distinction between sound conduction of the middle ear and sound conduction of the inner ear. The latter requires that the round window should not be exposed to the penetrating pressure of sound waves, a function normally performed by the tympanic membrane. In the absence of the latter, auditory prostheses, artificial membranes, etc., have been used, but these foreign bodies proved unsatisfactory. The aim of the intervention described by Moritz is to restablish sound conduction of the inner ear by producing two oscillating windows, one of which is protected against the pressure of sound waves. Before proceeding to the operation it is necessary to determine (1) the status of nervous function, (2) the status of sound conduction of the middle ear, (3) the mobility of the natural labyrinthine windows, and (4) the nature of the disease process involving the middle ear. The operation is only performed in the presence of large defects in the tympanic membrane, and two variants are described: (1) when both windows are functioning and (2) when the oval window does not vibrate. The plastic operation is combined with fenestration, whenever indicated. The described procedure results in marked auditory improvement, as demonstrated in a series of audiograms.

When acoustic energy is converted to mechanical energy the energy of the sound wave is divided into two streams of force of unequal pressure (Lempert [133]). The stream of greater pressure is directed along the ossicular chain against the oval window and the lesser stream against the round window. These streams of force are alternating in direction. The direction of thrust of the oval window is opposite in phase to that of the round window. These conclusions are supported by the general success of fenestration operation and by observation made on the effects of accidental perforations that occurred during fenestration.

Lavalle [134] reviewed a family of 104 members, with 24 having blue sclera. Of these, 20 had osteopsathyrosis and 16 were hard of hearing. The audiogram showed that the auditory defect was of the perceptive type and that the pure otosclerotic stage, if indeed it exists at all, is rapidly dominated by the cochlear affection. Hypoacusis commences at an age when fractures have ceased to occur.

Krassnig [135] reports experience in 1600 cases of trauma of the auditory nerve. Most of the patients were seen only after the injury had healed. On the strength of this material Krassnig regards the diagnosis of concussion of the labyrinth as justified, even though histologic proof is still lacking. Cerebral signs or symptoms are absent, but hearing is diminished to an equal degree throughout all ranges. As an analogy, reference is made to Berlin's edema of the retina (commotio retinae), which is due to concussion of the eyeball, a rigid structure filled with fluid and provided with sensory end-organs. In other instances it is necessary to assume the diagnosis of deafness due to brain stem injury. Neurologically, concussion of the brain stem is recognized and may result in permanent damage of the auditory organs. Up to the present time

Krassnig has encountered 15 cases which warrant this diagnosis.

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REFERENCES

1. Hall, I. S.: A New Approach to the Study of the Pathology of Otosclerosis, Acta oto-laryng., Supp. 100, pp. 164-168, 1952.

2. Nylen, C. O., and Nylen, B.: The Genesis of Otosclerosis, J. Laryng. & Otol. 66:55-64 (Feb.) 1952.

3. Brunner, H.: Pathology of Otosclerosis, A. M. A. Arch. Otolaryng. 55:369-380 (March) 1952.

4. Asherson, N.: Stapedial Fixation and Otosclerosis: The Differential Diagnosis, J. Laryng. & Otol. 66:552-569 (Nov.) 1952.

5. Cabanettes, J.: Practical Aspects of Atypical Forms of Otosclerosis, Rev. laryng. 73:247-253 (May-June) 1952.

6. Cawthorne, T.: Otosclerosis: A Review of Its Clinical Features as Noted in a Consecutive Series of 1150 Cases, Acta oto-laryng. 40:160-179, 1951 1952.

7. Rosen, S.: Palpation of Stapes for Fixation, A. M. A. Arch. Otolaryng. 56:610-615 (Dec.) 1952.

8. Hahlbrock, K. H.: Erweiterte Indikation zur Fensterungsoperation, Arch. Ohren- Nasen- u. Kehlkopfh. 161:435-440, 1952.

9. Shambaugh, G. E., Jr.: Pure Tone and Controlled Speech Tests in the Election of Cases for Fenestration, Acta oto-laryng. 40:199-210, 1951-1952.

10. Hall, I. S.: The Fenestration Operation and Its Results, Acta oto-laryng. 40:192-198, 1951-1952.

11. Shambaugh, G. E., Jr.: Present Status of the Technic of the Improved Fenestration Operation, Acta oto-laryng. 40:152-159, 1951-1952.

12. Hall, I. S.: Problems of the Fenestration Operation, J. Laryng. & Otol. 66:226-232 (May) 1952.

13. Rüedi, L.; Schneider, R.; Egli, P., and Perk, P.: Results of the Retro-Aural 2 Stages Fenestration Operation for Hearing Impairment Due to Otosclerosis, Acta oto-laryng. 40:226-237, 1951-1952.

14. Klotz, G.: Sourdille's Operative Method, Acta oto-laryng. 40:239-246, 1951-1952.

15. Garson, P.: Otosclerosis: A Closed Method of Fenestration, J. Laryng. & Otol. 66:65-70 (Feb.) 1952.

16. Loebell, G.: Zur Frage der Ausräumung der pneumatischen Zellen des Warzenfortsatzes bei der Fensterungsoperation, Ztschr. Laryng. Rhin. Otol. 31:147-150 (March) 1952.

17. Vyslonzil, E.: Die kontrollierte Blutdrucksenkung und ihre Bedeutung für gehörverbessernde Operationen (Fenestrationen), Wien. klin. Wchnschr. 64:772-775 (Sept. 26) 1952.

18. Goodhill, V.: The Use of Cortisone in Otosclerosis, Tr. Am. Acad. Ophth. 56:635-646 (July-Aug.) 1952.

19. Rambo, J. H. T.: Effect of Cortisone on Osteogenesis Following Fenestration in the Monkey: An Experimental Study, A. M. A. Arch. Otolaryng. 55:554-558 (May) 1952.

20. Lansberg, M. P.: Corticotropin (ACTH) and Fenestration, A. M. A. Arch. Otolaryng. 55: 662-665 (June) 1952.

21. Riskaer, N.: Response of Otosclerosis and Adhesive Otitis to Treatment with Corticotropin, A. M. A. Arch. Otolaryng. 55:343-349 (March) 1952.

22. Shambaugh, G. E., Jr.: Long Term Hearing Results in Fenestration Surgery, Acta oto-laryng. 40:180-191, 1951-1952.

23. Shambaugh, G. E., Jr.: Audiologic Assessment of Results in the Improved Fenestration Operation, Acta oto-laryng. 41:43-48, 1952.

24. House, H. P.: Long-Term Results of Fenestration Surgery, Ann. Otol. Rhin. & Laryng. 60:1153-1163 (Dec.) 1951.

25. Skoog, T.: The Transmission of Air Borne Sounds in the Fenestrated Ear in Clinical Otosclerosis, Acta oto-laryng., Supp. 100, pp. 134-143, 1952.

26. Nilsson, G.: The Immediate Improvement of Hearing Following the Fenestration Operation, Acta oto-laryng., Supp. 98, pp. 1-72, 1952.

27. McKenzie, W.: The Gain in Hearing After the Fenestration Operation and Its Influence upon the Surgeon's Choice of Cases, J. Laryng. & Otol. 66:211-225 (May) 1952.

28. Thullen, A.: Der Einfluss von Tuben- und Mittelohrkomplikationen auf das Gehör nach Fensterung, Arch. Ohren- Nasen- u. Kehlkopfh. 162: 126-134, 1952.

29. Zangemeister, H. E.: Über die Horbesserung des Gegenohres bei der Labyrinthfensterung, Arch. Ohren- Nasen- u. Kehlkopfh. 161:537-539, 1952.

30. Shambaugh, G. E., Jr.: Audiologic Studies of Failures, Acta oto-laryng. 40:211-218, 1951-1952.

31. Meltzer, P. E.: Indications of Revision of the Fenestration Opetration, Acta oto-laryng. 40: 219-225, 1951-1952.

32. Meurman, Y., and Savolainen, T.: Bacteriological Examination and Determination of Bacterial Resistance in Aural Suppuration Following Fenestration, Acta oto-laryng., Supp. 100, pp. 26-32, 1952.

33. Passow, H.: Das Verhalten des Vestibularapparates nach Fensterung, Ztschr. Laryng. Rhin. Otol. 31:351-359 (July-Aug.) 1952.

34. Meurman, Y., and Pursiainen, K.: The Caloric and Turning Tests After the Fenestration

Operation, Acta oto-laryng., Supp. 100, pp. 144-153, 1952.

35. Huizinga, E.: The Sound Reaction of Tullio in the Pigeon and the Fenestration Operation in Man, Acta oto-laryng., Supp. 100, pp. 174-180, 1952.

36. Huizinga, E.: The Reaction of Tullio and the Fenestration Operation, Laryngoscope 62:741-751 (July) 1952.

37. Lempert, J.; Wolff, D.; Rambo, J. H. T.; Wever, E. G., and Lawrence, M.: New Theory for the Correlation of the Pathology and the Symptomatology of Ménière's Disease, Ann. Otol. Rhin. & Laryng. 61:717-737 (Sept.) 1952.

38. Furstenberg, A. C.: Anatomical Considerations of Ménière's Disease, Ann. Otol. Rhin. & Laryng. 61:692-696 (Sept.) 1952.

39. Wildhagen, F.: Ménièresyndrome bei Affektionen der oberen Halswirbelsäule, Arch. Ohren-Nasen- u. Kehlkopfh. 159:242-246, 1951.

40. Meyer zum Gottesberge, A.: Das Hörbild der genuinen Ménièreschen Krankheit, Arch. Ohren- Nasen- u. Kehlkopfh. 159:402-406, 1951.

41. Fowler, E. P., Jr., and Zeckel, A.: Psychosomatic Aspects of Ménière's Disease, J. A. M. A. 148:1265-1268 (April 12) 1952.

42. Williams, H. L.: Allergy of the Inner Ear (Ménière's Disease), A. M. A. Arch. Otolaryng. 56:24-44 (July) 1952.

43. Richtner, N. G.: Some Aspects of Histamine in Ménière's Disease, A. M. A. Arch. Otolaryng. 55:434-443 (April) 1952.

44. Rosen, S.: Surgery and Neurology of Ménière's Disease: Role of Chorda Tympani Nerve in Tinnitus, Vertigo, and Deafness, A. M. A. Arch. Otolaryng. 56:152-160 (Aug.) 1952.

45. Frenckner, P.: Some Viewpoints on the Operative Treatment in Ménière's Disease, A. M. A. Arch. Otolaryng. 55:420-433 (April) 1952.

46. Day, K. M.: Symposium: Surgical Treatment of Hydrops of Labyrinth; Surgical Destruction of Labyrinth for Ménière's Disease, Laryngoscope 62:547-555 (June) 1952.

47. Flett, R. L.: Symposium: Surgical Treatment of Hydrops of Labyrinth; Portman Operation, Laryngoscope 62:556-561 (June) 1952.

48. Barber, H. O., and Ireland, P. E.: Symposium: Surgical Treatment of Hydrops of Labyrinth; Clinical Results Following Differential Section of Eighth Nerve, Laryngoscope 62:566-576 (June) 1952.

49. Krejci, F.: Experimentelle Grundlagen einer extralabyrinthären chirurgischen Behandlungsmethode der Ménièreschen Erkrankung, Pract. oto-rhino-laryng. 14:18-37, 1952.

50. Passe, E. R. G.: Sympathectomy in Relation to Ménière's Disease, Nerve Deafness and Tinnitus: Report on 110 Cases, Acta oto-laryng. 42:133-151, 1952.

51. Hoogland, G. A.: Treatment of Ménière's Disease with Cervical Sympathetic Block, Acta oto-laryng. 42:379-386, 1952.

52. Seiferth, L. B.: Zur Behandlung des Ménièreschen Syndroms (Ausschaltung des Nervus vestibularis durch Streptomycin), Ztschr. Laryng. Rhin. Otol. 31:484-487 (Oct.) 1952.

53. Cawthorne, T.: Streptomycin and Its Use in Ménière's Disease, Proc. Roy. Soc. Med. 45: 782 (Nov.) 1952.

54. Seymour, J. C., and Tappin, J. W.: The Effect of Sympathetic Stimulation upon the Cochlear Microphonic Potentials, J. Laryng. & Otol. 65:851-858 (Dec.) 1951.

55. Lindsay, J. R.; Schuknecht, H. F.; Neff, W. D., and Kimura, R. S.: Obliteration of the Endolymphatic Sac and the Cochlear Aqueduct, Ann. Otol. Rhin. & Laryng. 61:697-716 (Sept.) 1952.

56. Kristensen, H. K.: Acoustic and Vestibular Function in Guinea Pigs After Removal of the Membranous External Semicircular Canal, J. Laryng. & Otol. 66:259-276 (June) 1952.

57. Guild, S. R.: Conference on Problem of Noise in Industry: Ear Lesions Caused by Acoustic Trauma, A. M. A. Arch. Indust. Hyg. 5:121-123 (Feb.) 1952.

58. Gallagher, J. B., and Goodwin, J. E.: Temporary Changes in Acuity of the Human Ear Following Exposure to Intense Noise, A. M. A. Arch. Indust. Hyg. 6:332-338 (Oct.) 1952.

59. Rüedi, L.: Occupational Injuries of the Ear, Acta oto-laryng. 41:118-138, 1952.

60. Russbach, H.: Diagnostic Audiometry in Traumatic Deafness, Pract. oto-rhino-laryng. 14: 214-221, 1952.

61. Guild, E.: Acoustic Trauma in Aircraft Maintenance Workers, J. Aviation Med. 22:477-490 (Dec.) 1951.

62. House, H. P.: Conference on Problems of Noise in Industry: Clinical Aspects of Problems of Noise in Industry, A. M. A. Arch. Indust. Hyg. 5:124-128 (Feb.) 1952.

63. Korkis, F. B.: Blast Injuries of the Ear, J. Laryng. & Otol. 66:95-103 (Feb.) 1952.

64. Kryter, K. D.: Conference on Problems of Noise in Industry: Noise Safety Criteria, A. M. A. Arch. Indust. Hyg. 5:117-120 (Feb.) 1952.

65. Larsen, B.: Occupational Deafness: Clinic and Prophylaxis, Acta oto-laryng. 41:139-157, 1952.

66. Theilgaard, E.: Investigations in Auditory Fatigue in Individuals with Normal Hearing and in Noise Workers (Weavers), Acta oto-laryng. 39:525-537, 1951.

67. Sataloff, J.: Occupational Deafness, Indust. Med. 21:338-340 (July) 1952.

68. Boynton, W. D.: Noise and Noise Reduction, A. M. A. Arch. Indust. Hyg. 5:129-137 (Feb.) 1952.

69. Willemse, C.: Protection Against Occupational Deafness: Role of Vitamin A, Acta oto-rhino-laryng. belg. 6:319-323, 1952.

70. Cook, N.: Electrolytes and Noise Susceptibility, A. M. A. Arch. Otolaryng. 56:367-371 (Oct.) 1952.

71. Nash, C. S.: Industrial Loss of Hearing: Medical Aspects, Indust. Med. 21:171-173 (April) 1952.

72. Fox, M. S.: Industrial Noise: Medical, Economic and Social Aspects, Am. J. M. Sc. 223: 447-460 (April) 1952.

73. Mittermaier, R.: Ohrenärztliche Begutachtung unter besonderer Berücksichtigung der Erwerbsminderung, Arch. Ohren- Nasen- u. Kehlkopfh. 161:94-205, 1952.

74. Bauer, E.: Zur Problematik der otologischen Rentenbegutachtung, Arch. Ohren- Nasen- u. Kehlkopfh. 161:351-354, 1952.

75. Symons, N. S.: Conference on Problems of Noise in Industry: Legal Aspects of Occupational Deafness, A. M. A. Arch. Indust. Hyg. 5:138-156 (Feb.) 1952.

76. Carter, H. A.: Conference on Problems of Noise in Industry: Estimating Percentage Loss of Hearing, A. M. A. Arch. Indust. Hyg. 5:113-116 (Feb.) 1952.

77. Walker, A. S.: Hearing of Patients After Tuberculous Meningitis, Proc. Roy. Soc. Med. 45:781-782 (Nov.) 1952.

78. Hawkins, J. E., and Lurie, M. H.: The Ototoxicity of Streptomycin, Ann. Otol. Rhin. & Laryng. 61:789-809 (Sept.) 1952.

79. Whetnal, E. M., and Lucas, H. A.: Changes in Internal Ear and Eighth Nerve in Fatal Cases of Tuberculous Meningitis Treated by Streptomycin and Dihydrostreptomycin, Proc. Roy. Soc. Med. 45:779-781 (Nov.) 1952.

80. Rüedi, L.; Furrer, W.; Luthy, F.; Nager, G., and Tschirren, B.: Further Observations Concerning the Toxic Effects of Streptomycin and Quinine on the Auditory Organ of Guinea Pigs, Laryngoscope 62:333-351 (April) 1952.

81. Schwab, W.: Akustikusausfall und Tetanus (Infektionsfolge oder Serumschädigung), Ztschr. Laryng. Rhin. Otol. 31:368-371 (July-Aug.) 1952.

82. Wullstein, H.: Schwerhörigkeit in Abhängigkeit von einem Focus? Ztschr. Laryng. Rhin. Otol. 31:541-545 (Nov.) 1952.

83. Cawthorne, T.: Vertigo, Brit. M. J. 2:931-933 (Oct. 25) 1952.

84. Mygind, S. H.: Function and Diseases of the Labyrinth, Acta oto-laryng. 41:235-321, 1952.

85. Dix, M. R., and Hallpike, C. S.: The Pathology, Symptomatology and Diagnosis of Certain Common Disorders of the Vestibular Mechanism, Ann. Otol. Rhin. & Laryng. 61:987-1016 (Dec.) 1952.

86. Lindsay, J. R.: Postural Vertigo and Positional Nystagmus, Ann. Otol. Rhin. & Laryng. 60:1134-1152 (Dec.) 1951.

87. Wodak, E.: Diagnostic Value of the Latency Period After Caloric Stimulation of Vestibular Apparatus, A. M. A. Arch. Otolaryng. 55:381-386 (March) 1952.

88. Vilstrup, G., and Vilstrup, T.: Does the Utricular Otolithic Membrane Move on Postural Changes of the Head? Ann. Otol. Rhin. & Laryng. 61:189-197 (March) 1952.

89. Jongkees, L. B. W.: Some Remarks on Vestibular Examination, Proc. Roy. Soc. Med. 45:127-133 (March) 1952.

90. Lowenstein, O.: Recording of Responses from Individual End-Organs of the Vestibular Apparatus, Proc. Roy. Soc. Med. 45:133-134 (March) 1952.

91. Boenninghaus, H. G.: Die Beeinflussung des experimentell ausgelösten Nystagmus und Schwindels durch Vomex A, Ztschr. Laryng. Rhin. Otol. 31:558-564 (Nov.) 1952.

92. Symonds, C.: The Significance of Vertigo in Neurological Diagnosis, J. Laryng. & Otol. 66:295-301 (July) 1952.

93. Lewis, R. S.: Sympathectomy and the Internal Ear, J. Laryng. & Otol. 65:825-842 (Dec.) 1951.

94. Lauter, S.: Leitsymptom: Schwindel, München. med. Wchnschr. 94:537-544 (March 21) 1952.

95. Hirsh, I. J.: Audiology and the Basic Sciences, Acta oto-laryng. 40:42-50, 1951.

96. Metz, O.: Threshold of Reflex Contractions of Muscles of Middle Ear and Recruitment of Loudness, A. M. A. Arch. Otolaryng. 55:536-543 (May) 1952.

97. Tiffany, W. R., and Hanley, C. N.: Delayed Speech Feedback as Test for Auditory Malingering, Science 115:59-60 (Jan. 18) 1952.

98. Kristensen, H. K., and Jepsen, O.: Recruitment in Otoneurologic Diagnosis, Acta oto-laryng. 42:553-560 (Dec.) 1952.

99. Jerger, J. F.: Difference Limen Recruitment Test and Diagnostic Significance, Laryngoscope 62:1316-1332 (Dec.) 1952.

100. Huizing, H. C., and Reyntjes, J. A.: Recruitment and Speech Discrimination Loss, Laryngoscope 62:521-527 (May) 1952.

101. Watson, T. J.: Ascertainment of the Need for Special Educational Treatment in Children of School Age with Defective Hearing, Acta oto-laryng. 40:361-369, 1951-1952.

102. Watson, T. J.: Auditory Training and the Development of Speech and Language in Children wih Defective Hearing, Acta oto-laryng. 40:95-103, 1951.

103. Hardy, W. G.; Pauls, M. D., and Bordley, J. E.: Modern Concepts of Rehabilitation of Young Children with Severe Hearing Impairment, Acta oto-laryng. 40:80-86, 1951.

104. Bergman, M.: Principles and Basic Techniques of Adult Auditory Rehabilitation, Acta oto-laryng. 40:324-335, 1951-1952.

105. Bergman, M.: Special Methods of Audiological Training for Adults, Acta oto-laryng. 40: 336-345, 1951-1952.

106. Dix, M. R., and Hallpike, C. S.: Further Observations on Diagnosis of Deafness in Young Children, with Particular Reference to the Making of Recommendations for Special Educational Treatment, Brit. M. J. 1:235-244 (Feb. 2) 1952.

107. Hall, I. S.: The Prevention and Treatment of Deafness in School Children, Acta oto-laryng. 40:87-94, 1951.

108. Carson, E. T.: Practical Aspects of Unilateral and Moderate Hearing Losses, A. M. A. Arch oto-laryng. 55:525-527 (May) 1952.

109. Gerrard, J.: Nuclear Jaundice and Deafness, J. Laryng. & Otol. 66:39-46 (Jan.) 1952.

110. Johnsen, S.: Natal Causes of Perceptive Deafness, Acta oto-laryng. 42:51-63, 1952.

111. Johnsen, S., and Freiesleben, E.: The Relation Between Erythroblastosis Foetalis, Kernicterus and Impairment of Hearing, Acta oto-laryng. 42: 35-50, 1952.

112. Lancaster, H. O.: Deafness as an Epidemic Disease in Australia: A Note on Census and Institutional Data, Brit. M. J. 2:1429-1432 (Dec. 15) 1951.

113. Nager, F. R.: Histologische Ohruntersuchungen bei Kindern nach mütterlicher Rubella, Pract. oto-rhino-laryng. 14:337-359, 1952.

114. Novotny, O.: Die Altersschwerhörigkeit, Wien. klin. Wchnschr. 64:458-459 (June 20) 1952.

115. Kobrak, F.: Altersschwerhörigkeit, eine Schwäche des "Hörtonus" (Hypotonia senilis acustica), Ztschr. Laryng. Rhin. Otol. 31:232-235 (May) 1952.

116. Saxen, A.: Inner Ear in Presbyacusis, Acta oto-laryng. 41:213-227, 1952.

117. Johnsen, S.: The Heredity of Perceptive Deafness, Acta oto-laryng. 42:539-552, 1952.

118. Meyer zum Gottesberge, A.: Zur funktionellen Pathologie der Innenohrschwerhörigkeit, Arch. Ohren- Nasen- u. Kehlkopfh. 161:546-548, 1952.

119. Bordley, J. E., and Hardy, W. G.: The Etiology of Deafness in Young Children, Acta oto-laryng. 40:72-79, 1951-1952.

120. Parlman, H. B., and Leek, J. H.: Late Congenital Syphilis of the Ear, Laryngoscope 62: 1175-1196 (Nov.) 1952.

121. Phillips, J. W.: Otologic Symptoms in Multiple Sclerosis, Laryngoscope 62:271-276 (March) 1952.

122. Asher, P.: Study of 63 Cases of Athetosis with Special Reference to Hearing Defects, Arch. Dis. Childhood 27:475-477 (Oct.) 1952.

123. Brown, H. A.; Love, J. G., and Adams, N. D.: Otologic Evaluation of Unilateral Acoustic Neurofibroma, Laryngoscope 62:250-261 (March) 1952.

124. Engström, H., and Graf, W.: Recording of Objective Tinnitus, Acta oto-laryng. 41:228-234, 1952.

125. Baron, S. H.: Experiences with Parenteral Vitamin A Therapy in Deafness and Tinnitus, Tr. Pacific Coast Oto-Ophth. Soc. 32:71-94, 1952.

126. Lawrence, M., and Wever, E. G.: Effects of Oxygen Deprivation upon the Structure of the Organ of Corti, A. M. A. Arch. Otolaryng. 55:31-37 (Jan.) 1952.

127. Portmann, G.; Portmann, M., and Barbe, L. J.: Experimental Investigations on the Effect of Supersonic Waves on Hearing, Acta oto-laryng., Supp. 100, pp. 119-133, 1952.

128. Schuknecht, H. F., and Neff, W. D.: Hearing Losses After Apical Lesions in the Cochlea, Acta oto-laryng. 42:263-274, 1952.

129. Zöllner, F.: Plastische Eingriffe an den Labyrinthfenstern, Arch. Ohren- Nasen- u. Kehlkopfh. 161:414-422, 1952.

130. Shambaugh, G. E., Jr.: Developmental Anomalies of the Sound Conducting Apparatus and Their Surgical Correction, Ann. Otol. Rhin. & Laryng. 61:873-887 (Sept.) 1952.

131. Pattee, G. L.: Improved Hearing in Congenital Auricular Malformations: Review of Results Obtained by Surgical Methods, Tr. Am. Acad. Ophth. 56:465-474 (May-June) 1952.

132. Moritz, W.: Plastische Eingriffe am Mittelohr zur Wiederherstellung der Innenohrschalleitung, Ztschr. Laryng. Rhin. Otol. 31:338-351 (July-Aug.) 1952.

133. Lempert, J.: Physiology of Hearing: What Have We Learned About It Following Fenestration Surgery? A. M. A. Arch. Otolaryng. 56: 101-113 (Aug.) 1952.

134. Lavalle: The Syndrome of Blue Sclerotics (van der Hoeve Syndrome), Acta oto-rhino-laryng. belg. 6:379-396, 1952.

135. Krassnig, M.: Die Begutachtung posttraumatischer Ohrschaden ohne örtlichen Befund, Arch. Ohren- Nassen- u. Kehlkopfh. 161:325-329, 1952.

Abstracts from Current Literature

Ear

Treatment of Infected Ears with Oxytetracycline (Terramycin)-Polymyxin. J. R. Ausband, Eye Ear, Nose & Throat Month. 34:746 (Nov.) 1955.

A combination of oxytetracycline (Terramycin) and polymyxin B was used in the treatment of both otitis media and otitis externa. The mixture was found to be very effective in cases of otitis externa, but to be of little benefit to a small group of patients with chronic purulent otitis media.

Jennes, Waterbury, Conn.

Audiologic Examination of the Inner Ears: The Aural-Overload Test. Phillip A. Yantis, J. Speech & Hearing Disorders 21:303 (Sept.) 1956.

Audibility thresholds are obtained for the fundamental tone to be used and for the second harmonic. The fundamental frequency is then set on one pure-tone oscillator so that it will be 70 db. above the audibility threshold for that tone. On a second oscillator the exploring-tone frequency is made to differ from the frequency of the second harmonic by 4 cps and adjusted to 60 db. above threshold. The tones are then mixed and switched to the subject's ear, and attenuated until beats are no longer heard. Inner-ear pathology is demonstrated by abnormally restricted range.

Palmer, Wichita, Kan.

The Adenoid and Its Relation to the Ear. Lucian W. Alexander, Laryngoscope 66:177 (June) 1956.

This article reviews the fairly well-known facts concerning the adenoids. This tissue should be removed only if it is hypertrophied and is causing obstruction in breathing or to the Eustachian tubes, or if it is chronically diseased. If diseased, palpation usually reveals the finger covered with tenacious mucus streaked with blood. Conductive auditory impairment and chronic otitis media are, of course, positive indications for adenoid removal. A complete removal is essential. Inspection of the fossae of Rosenmüller may reveal unremoved tissue there. A punch forceps is useful for this. X-ray and/or radium treatment should be reserved for obstructive adenoidal tissue in the Eustachian tubes. X-ray is preferred for the very young and radium for the older child.

Hitschler, Philadelphia.

Practical Office Audiometry. Wesley H. Bradley, New York J. Med. 56:2975 (Oct. 1) 1956.

The field of otology has greatly widened in recent years and abounds in new terminology, such as recruitment, difference limen, spondee words, etc. "The average otologist . . . attempting to render the most complete service . . . may often question whether he is achieving this result." How may we achieve this practically in our clinical or office work? Pure-tone audiometry for both bone and air conduction is dependent on the audiograph machine, but tuning forks may, or perhaps should also be used. In any case the examiner and examinee may not "click," and different readings may be taken by different examiners, with a percentage of error likely. The "personal equation" has something to do with the matter. Masking is important, but "at the present time there is no ideal medium for masking." White noise is helpful, and some audiometers now have this item built into their mechanism. The human voice is, of course, the sound we most desire to hear, but it is difficult to standardize because of so many variations. As for pure tone, a person may do quite well in the test yet have difficulty hearing his associates. "There is no really good, reliable recorded test material commercially available for speech audiometry." Not too much should be left to an assistant; if live voice is preferred, the examiner himself should make the tests. Such testing may be helpful in the following instances: 1. In prefenestration, for the operator who is preparing to do fenestration or mobilization feels more secure if he knows that the patient has a discrimination score of 90% or above. 2. In establishing Ménière's data, speech testing may be very helpful. The

loss for pure tones may not be great, but the discrimination score is always very poor. 3. From a very practical view, the otologist is better able to advise the patient as to a hearing aid after speech testing. 4. Pure tone plus speech is often needed to advise and counsel a patient who has an irreversible loss. This discussion period with the patient is of primary value, no matter what the status. He leans on us for our opinion of his present hearing loss, the cause (if known), and the remedy (if any).

ABSTRACTER'S COMMENT: We sometimes may be obtuse to the fact that one hears not with the ears but with the brain! Deafness may be both conductive and perceptive in the same patient. Or conduction may seem not to be greatly interfered with, but interpretation may be curiously lacking or far removed from normal. There are probably some people whose amount of hearing cannot be evaluated by tests, and there are certainly many who cannot be fitted with an aid. The aid may be excellent, but the use of it is entirely subjective and may fail a given user almost completely.

I. W. VOORHEES, New York.

A HISTOLOGIC STUDY OF THE ACTION OF HYALURONIDASE ON THE INTERNAL EAR. JAIME MARCO, HERMINIO MORERA, AND JOSÉ-ANTONIO GIMÉNEZ, Acta oto-laryng. 46:398 (Sept.-Oct.) 1956.

A histophysiologic investigation was performed on the labyrinth of the guinea pig. In some hyaluronidase was injected into the tympanic cavity. This was done through a puncture in the tympanic membrane. The purpose of the experiment was to see what this enzyme would do in effect on reaching the internal ear and the labyrinth.

Functional studies and histological examination failed to show any abnormality of ears treated with hyaluronidase when compared with normal untreated ears. The authors quote a personal communication from Vilstrup, who made the same findings.

LEWY, Chicago.

THE FUNCTIONAL EXAMINATION OF THE EAR IN CHRONIC OTITIS MEDIA WITH THE AID OF ACOUSTIC SOUND. R. A. DEPRIEST AND J. DEPAEPE, Acta oto-rhino-laryng. belg. 10:20, 1956.

The authors have used a special acoustic sound which is connected to an audiometer and placed either on the promontory, the malleus, or on the incudostapedial articulation. Audiograms are taken at these areas, and from these interpretations of various forms of deafness can be made. Furthermore, one can evaluate on the basis of these findings the type of surgery that is indicated and whether or not there will be any postoperative improvement in the hearing acuity. They believe that if there is a hearing loss from zero to 20 db. one should conserve the ossicular chain. If the hearing loss is between 20 and 40 db., they advise a conservative operation (with perhaps the removal of the incus and the malleus and preservation of the tympanum) or doing a tympanoplasty. If the loss is over 40 db. a complete radical operation is indicated. Here they frequently apply a plastic plug to cover the stapes.

PERSKY, Philadelphia.

THE TREATMENT OF OBSTRUCTIVE DEAFNESS FOLLOWING CHRONIC SUPPURATIVE OTITIS MEDIA. JUAN MANUEL TATO AND GONZALO DE SEBASTIÁN, Acta oto-rino-laryng. Ibero-am. (Barcelona) 7:309, 1956.

The authors discuss obstructive deafness in a series of otitic conditions: 1. Where there is no perforation of the tympanum nor adhesions between the tympanum and the promontory. This type is usually found in secretory otitis. The diagnosis is easily established, and the treatment is either catheterization, protein therapy, or the use of cortisone or corticotropin (ACTH). 2. Central perforations of the tympanum without alteration of the ossicular chain and without epithelization of the tympanic cavity. Here usually one finds a hearing loss of 15 to 30 db. The diagnosis is very simple—either by simple otoscopy, the audiogram, the sound of Zollner, or the audiometric result following the use of an artificial tympanum. 3. Marginal perforation without alteration of the ossicular chain or without epithelization of the tympanic cavity. Otologically the diagnosis is similar to that in Group 2. 4. Tympanic perforation with adhesions to the promontory, with or without epithelization of the tympanic cavity. 5. Marginal or central perforations with lesions of the ossicular chain and without epithelization of the middle ear. Here usually the diagnosis is very easy, but the treatment is varied. 6. Postoperative mastoidectomies with a sequela which involves all the previous groups.

In analyzing the various pathologic conditions, the following conclusions can be made: 1. An electrical prosthesis is extremely useful in these patients. 2. In regard to a mechanical prosthesis, artificial eardrums can be made of cellophane, rubber, and various types of plastics. 3. Cauterization of the edges of the perforation with silver nitrate or trichloroacetic acid has produced excellent results in a considerable number of patients. 4. Tympanoplasty performed after surgery on a mastoid process has not only improved the hearing but has also improved the pathologic process underlying the loss of hearing. 5. Fenestration is reserved for those cases where there is a loss of over 60 db. In conclusion, the authors believe that a most careful study of the underlying pathology is indicated. This would direct them toward the proper type of therapy to be used.

PERSKY, Philadelphia.

FUNCTIONAL SURGERY OF CHRONIC OTITIS MEDIA. M. PRUVOT, Rev. laryng. (Bordeaux) 77: 261 (March-April) 1956.

The author discusses the various pathologic conditions occurring in dry otitis media with the thought of improving audition. As an indication for his therapy he describes five types of perforations: (1) the small- or middle-sized perforation of the middle of the tympanum, (2) large mesotympanic perforation, (3) medium posterior epitympanic perforation, (4) medium anterior epitympanic perforation, and (5) large epitympanic perforation. For each group there is more or less a different method of treatment. In Group 1, he merely freshens up the edges of the perforation and applies a small skin graft to the area. This is held in place with a little pledget of petrolatum (Vaseline) cotton. In the second group he uses a larger skin graft but may have to insert an absorable packing in the middle ear to act as a buffer between the flap and the promontory to prevent adhesions. In the third group he frees the skin according to a Rosen incision and the flap is placed both on the tympanum and on the bony surface. In Group 4, the procedure is similar to that for Group 3. However, in Group 5 there usually is associated a considerable amount of pathology, not only in the middle ear but also involving the ossicular chain. Here tympanoplasty according to the technique of Wullstein is performed. This varies according to the underlying pathology, namely, pathology involving (1) the ossicules, (2) the oval window, (3) the round window, (4) the hypotympanum, and (5) the orifice of the Eustachian tube. In simple pathology involving the ossicular chain without fixation of the footplate of the stapes, the malleus and incus may be removed. Where the stapes has been fixed by fibrosis, this can be freed by the Rosen technique. The hypotympanum and the region of the orifice of the Eustachian tube can be cleared up and the flap can be inserted throughout the area, with excellent results. In some instances where the pathology involves the round window or the oval window an attempt can be made to remove the pathology and fibrosis. Care must be exercised so as not to penetrate the round window or dislodge the footplate of the stapes. A skin graft is applied, and absorbable gelatin holds it firmly in its bed. The dressing is removed on the 10th day. This procedure has given results equal to that of the more extensive procedure of exposing the entire tympanic cavity. Furthermore, the operative time is shortened, there is less hemorrhage, less postoperative care, and less trauma and shock to the patient.

PERSKY, Philadelphia.

Larynx

MECHANICAL HEAD SUPPORT FOR BRONCHOESOPHAGOLOGY. ALBERT H. ANDREWS JR. and ARTHUR LOEWY, Eye, Ear, Nose & Throat Month. 35:38 (Jan.) 1956.

In a timely article the authors discuss the problems attending the performance of bronchoesophagologic procedures in the absence of experienced or adequate personnel. In the evaluation of mechanical means of maintaining head position, the head rest designed by Haslinger in the early 1920's most closely fulfills the requirements. It is easily attached to an operating table and supports the head adequately. It can be maintained in a head-high position, with the head at the level of the table, or below the horizontal plane of the table, and side-to-side motion is possible in any of the vertical positions. The control of these changes of position is readily available to the operator. The practical limitations of the use of this head rest are dependent upon the size of the head rest, which makes it difficult to use in a person less than 5 ft. tall. It may be used in anyone old enough to be cooperative.

ABSTRACTER'S COMMENT: In many a hospital a head rest such as the Haslinger is hidden away in some corner of a storage closet. Considering the virtual disappearance of the intern

from the bronchoscopic operating room and the difficulty in maintaining trained nonmedical head holders in this day of rapid turnover of personnel, it behooves us all to unearth the neglected mechanical head holder and put it to work. How different from the days when in each bronchoesophagologic procedure there was, in addition to the experienced operator, an experienced head holder and a well-trained shoulder holder!

JENNES, Waterbury, Conn.

Nose

RADIATION THERAPY FOLLOWING RHINOPLASTY. SIDNEY M. SILVERSTONE, Eye, Ear, Nose & Throat Month. 34:735 (Nov.) 1955.

After a rhinoplasty there is usually a postoperative reaction with swelling of the tip and dorsum of the nose. This is due to a subcutaneous edematous inflammatory reaction with some ecchymosis. This generally disappears within a few weeks. In some patients there is a persistent swelling which is due both to a moderate amount of nonresorbed subcutaneous edema and, at times, to a thickening of the skin itself. The skin is coarse with enlarged pores and an irregular surface. This represents an abnormal method of healing peculiar to the person and is common in patients who have acne or seborrheic dermatitis or who are subject to keloid or hypertrophic scar formation. The skin changes may take many months to clear or may be permanent. Radiation therapy has been of value in dermatologic cases with a similar form of thickening of the epidermis and dermis.

Radiation therapy should therefore be of value in cases of rhinoplasty with this same type of problem. Silverstone describes in detail his technique for radiation therapy following rhinoplasty, which may be used with safety and without risk of late sequelae. The face, with the exception of the nose, is shielded adequately with lead. Radiation generated at 200 kv. is employed with a Thoraeus filter and a target-skin distance of 50 cm. Six treatments of 50 r (in air) spaced a day or two apart are given. The total dose, therefore, is 300 r (in air), and is completed within 6 to 10 days.

Treatment is usually begun as soon as the process is recognized, which is generally between one to four weeks after the operation. Improvement is generally noticeable from one to three weeks after the last treatment. The therapeutic response in these patients with bulbous enlargement of the nose consists of a gradual disappearance of the edema and a restoration of the skin to its normal smooth texture. This form of treatment may be used prophylactically to good advantage when it can be suspected from the nature of the skin that abnormal healing will ensue. Treatment is then administered from one to two weeks following rhinoplasty.

JENNES, Waterbury, Conn.

MESIAL CRURA: IMPORTANCE IN NASAL TIP RECONSTRUCTION. IRVING B. GOLDMAN, Eye, Ear, Nose & Throat Month. 34:753 (Nov.) 1955.

Adequate surgery of the nasal tip is of the utmost importance in good rhinoplasty. Excessive resection of cartilage, vestibular skin, or mucosa results in an unpleasant looking and poorly functioning nose. Untoward sequelae include alar cleft, alar collapse, vestibular stenosis, and the familiar "pinched-in" surgical appearance. A multitude of sutures and techniques of suturing have been described to avoid these undesirable effects. Overcorrection, the use of battens and buttons, and prolonged dressings have all been used to try to prevent postoperative drooping of the nasal tip.

The surgical anatomy of the lower lateral cartilages is described in detail. A technique is described by which a satisfactory nasal tip may be obtained without the use of special sutures, battens, or buttons. A circumferential rim incision is made within the lower margins of the nostrils, including the posterior part of the columella for about one-fourth of its length. The soft tissues of the tip are separated, including the lobule. The lower lateral cartilages are exposed and the angles cut. The mesial crura are approximated by several absorbable surgical (gut) sutures, and the conjoined mesial crura are angulated so that the caudal margin is higher than the cephalic margin. The important step in this technique consists of forming a straight caudal margin of the septum instead of the normal septal curve. The straight caudal margin of the septum when sutured to the mesial crura by septocolumellar sutures acts as a batten to support the mesial crura. Nasal tips treated in this fashion exhibit unusual firmness, which is exceedingly helpful in contouring the nasal tip.

JENNES, Waterbury, Conn.

PSYCHIATRIC PROBLEMS IN RELATION TO RHINOPLASTY. IRVING B. GOLDMAN, Eye, Ear, Nose & Throat Month. 35:128 (Feb.) 1956.

In a very worthwhile addition to his monthly comments on plastic surgery, Goldman describes the "rhinoplastic syndrome." His description of the typical patient demonstrating psychic distress because of nasal deformity should be read, particularly by those who do not do rhinoplastic surgery. The rhinoplastic surgeon encounters this psychic symptom complex so frequently that it becomes commonplace. Much of the rewards and pleasures of performing such surgery is in seeing the transformation of these people following adequate rhinoplastic surgery. The change in psychic outlook is rapid and remarkable. These patients are among the most appreciative patients a physician may ever know.

The nose plays a special role in the concept of the body image. When this is not personally pleasing, the person is psychiatrically ill. With the correction of the nasal deformity, a pleasing body image is restored, with resulting improvement in mental or psychic health. Alteration in the direction of greater conformity to the social average is also helpful to the restoration of psychic health in this type of patient.

The disruption in the patient's life frequently seems disproportionate to the degree of actual nasal deformity. A small but real nasal deformity can, however, cause marked though seemingly disproportionate symptoms. Psychic energy is immobilized by the patient's preoccupation with his nose. The relief obtained from operation is measurable therefore not by the size of the deformity but by the quantity of psychic energy not utilized. The patient has an increased store of energy available for utilization in effective adjustments to life situations. Psychosis is a rare sequel of rhinoplasty. A successful rhinoplasty is not a hazardous procedure from the psychiatric point of view, and frequently is important in the therapy of the patient with severe psychiatric problems. He may still have problems, but he is more amenable psychotherapeutically after operation than he was preoperatively. Close cooperation between plastic surgeon and psychiatrist will bring about improved psychotherapeutic results and continued progress in psychotherapeutic theory.

JENNES, Waterbury, Conn.

Pharynx

HAEMOPHILIA INVOLVING THE PHARYNX AND LARYNX. B. H. COLMAN, J. Laryng. & Otol. 70:540 (Sept.) 1956.

This article is a case report of a relatively rare complication of hemophilia, and a discussion of the specific complication. The case was that of a known hemophiliac who had had various hemorrhages in the past, and came in with a severe hematoma involving the peritonsil area of the pharynx and dissecting down into the hypopharynx. The false vocal cords were quite edematous and blue. The patient was in considerable respiratory distress, and a very careful decision was required concerning tracheotomy. Tracheotomy was not done. Antihemophilic plasma was used along with local measures, and the hematoma and reaction subsided very rapidly without further intervention. A brief discussion of hemophilia, as it may involve the air and food passages, is presented.

LEJEUNE AND STEVENSON, New Orleans.

Miscellaneous

THE CLINICAL APPLICATION OF BIOFLAVONOIDS IN OTOLARYNGOLOGY. HERBERT B. GOLDMAN, Eye, Ear, Nose & Throat Month. 35:246 (April) 1956.

Bioflavonoids were administered in 530 tonsillectomy cases, 35 surgical cases other than tonsillectomies, and in 47 patients with recurrent nose bleeds. Bioflavonoids control capillary permeability and fragility. Medication was given for five to eight days prior to and four to five days following surgery. Operative hemorrhage was decreased and subsequent oozing was lessened. In the patients with recurrent epistaxis who were maintained on the bioflavonoids for a period of from several weeks to months, there was a sharp decrease in the number of nose-bleed recurrences.

ABSTRACTER'S COMMENT: It is unfortunate that it is so difficult to provide quantitative reports on the action of new drugs, especially when operative and postoperative bleeding is involved.

Evaluation of such therapy would be benefited by the use of controls and by the quantitative estimation of blood loss. The blood aspirated into the suction bottle should be measured, and operation should be performed with the head hyperextended to prevent blood loss by swallowing.

JENNES, Waterbury, Conn.

ADENOTONSILLECTOMY IN THE ALLERGIC CHILD. GERTRUDE RUSSACK SOBEL, New York J. Med. 56:3172 (Oct. 15) 1956.

Dr. Sobel gives her experiences based upon service in the Meadowbrook Hospital, Hempstead, N. Y. Anxious parents often inquire concerning what effect adenotonsillectomy will have on "my child's allergy. Three answers are at hand: The allergy will improve; it will get worse; no difference will be manifest. There are also three schools of thought concerning adenotonsillectomy: 1. Adenoids and tonsils often are foci of infection; therefore removal is in order. 2. These organs are there for a purpose, very likely to offer a first line of defense against germs. Removal may intensify allergy, may convert a mild asthma into a vicious one. 3. Indications for operation are the same in nonallergic and allergic patients. Chronically diseased or abnormally enlarged tonsils should be enucleated. It is well to put through a course of antiallergic agents, then wait and see if surgery is necessary. 4. Surgical nihilists are against removal of these organs. They aim to eradicate infection through antibiotics, allergic management, general physical build-up, aerosols, etc. No statistical difference has been found in patients operated on and those not operated on as to asthma or allergy *en grosse*. Six principles are now generally accepted: Do adenotonsillectomy in chronic infected cases, in abnormal enlargement of the tonsil which interferes with swallowing and breathing, in persistent cervical adenitis, in recurrent otitis media, in diminished hearing from nasopharyngeal pathology, and in repeated upper respiratory infections with cardiac and renal complications.

A contraindication to A. and T. removal is the time of year. No operation should be performed during the pollen season: April through October. Ill timing may favor the establishment of asthma. Pending decision for surgery, one should treat the infection vigorously, determining sensitivity, specific antibodies, cultures and smears, and specific food elimination. Some 40% of the patients seen in an allergist's office have already experienced A. and T. removal. When performed against allergy, success was not common. Allergists find that only 4-8% require surgery if medical care has been incisive and thorough. Moreover, allergic patients are "prone to infection." Altered function of the mucous membrane sets the stage for germ invasion; therefore allergy and secondary infection may and often do coexist. When regrowth takes place after surgery one may suspect an underlying allergy. Some 7% of the patients seen by the allergist have had more than one adenotonsillectomy! Lymphoid hyperplasia in allergy is almost the rule, especially in the nasopharynx, and too often the allergic base is overlooked. Differential diagnosis between allergy and infection may be difficult, but nasal smears with an eosinophile count of 4% or more are suspect for allergy. With infection, one is likely to find polymorphonuclears predominant, with or without eosinophiles. In summary, "There is often no need for adenotonsillectomy in the allergic patient after successful antiallergic and anti-infective therapy." If A. and T. surgery becomes imperative, it should be done out of the pollination season, not during. "The presence or absence of tonsils or adenoids has no bearing on the basic allergic pattern."

ABSTRACTER'S COMMENT: It is doubtless true that many infections may be cleared up by present-day management without surgery. The chief point is not to delay surgery too long (e. g., in mastoiditis). It will surprise some A. and T. men to learn that surgery may worsen allergies. As for tonsil function, compare it to a coal sieve. The sieve is very useful in holding back clinkers, but when every crevice is filled with them, the sieve is not longer useful.

I. W. VOORHEES, New York.

DISCUSSION ON UNUSUAL ASPECTS OF ESOPHAGEAL DISEASE. N. R. BARRETT, Proc. Roy. Soc. Med. 49:529 (Aug.) 1956.

This is a report of the meeting of the Section of Laryngology held on Feb. 3, 1956, under the presidency of W. A. Mill, M.S., F.R.C.S. Instrumentation (esophagoscopy) is set down as a more or less common cause of perforations! Then follow sharp foreign bodies, stab wounds of the neck, pathological lesions of the gullet, and spontaneous rupture from excessive vomiting. Two areas are vulnerable: (1) the neck, cervical esophagus; (2) the lower esophagus. Twenty-two cases in 15 years are set down in tabulated form. The majority were seen late in their disability; therefore great stress is placed on early diagnosis and prompt surgery.

Pharyngeal perforations are commonly due to misadventures in instrumentation during preparations for anesthesia. Here surgical emphysema will ensue as soon as the gaseous anesthetic is begun. "The situation is sudden and critical." In such a case, stop the gas, insert an intratracheal tube to maintain respiration, and then deflate the neck by incisions through the deep fascia above the clavicle. Cervical perforations are relatively common and lie below the cricopharyngeal sphincter in the midline of the gullet posteriorly. It is seen in old persons chiefly, and may not be manifest for two or three days after instrumentation. In all cases the "expectant method courts danger." The following generalizations were made: 1. Perforations of the cervical esophagus are seldom diagnosed early. Symptoms and signs are likely to be delayed. 2. Use of antibiotics alone is a hazard because of a disastrous waiting period. 3. Lateral radiographs of the neck may bring positive diagnosis. 4. The surgeon must expose the esophagus at once, repair the damage, establish temporary drainage, and administer antibiotics. 5. To await developments before performing surgery is to risk the development of mediastinal abscess, with possible pyopneumothorax. 6. Late drainage may possibly save the patient's life, but it carries a high risk of fatality and chronic illness. Suppurative thoracic complications are worrisome, time-consuming, and extend over weeks of hospitalization.

Rupture from vomiting causes a longitudinal tear, 1 to 2 cm. in length, which is situated just above the diaphragmatic hiatus. Gastric contents seep into the mediastinum or through the pleura into the lungs, causing a "chemical mediastinitis," the principal feature of which is "hemorrhagic necrosis." Spontaneous perforation of the esophagus is a lethal condition, formerly fatal; but due to newer methods of diagnosis and more expert surgical technology, a good percentage of uneventful recoveries are coming to light. The author quotes Wallace, who said: "It is safer to look and see, than to wait and see."

ABSTRACTER'S COMMENT: Here is the most exhaustive study of this important surgical subject to be seen in the literature for many years.

I. W. VOORHEES, New York.

DISCUSSION ON CANCER RESEARCH: ITS PRESENT TRENDS. A. HADDOW, G. HADFIELD, P. R. PEACOCK, Procs. Roy. Soc. Med. 49:661 (Sept.) 1956.

This is a report of a symposium held on March 2, 1956, with President W. A. Mill, M.S., F.R.C.S., in the chair. Professor Haddow works at the Chester Beatty Research Institute, Institute of Cancer Research, Royal Cancer Hospital. His theme was "The Study of Carcinogenesis." Some 40 years ago the Japanese began the experimental study of carcinoma induced by applying coal tar to the ears of rabbits. The effort was made to determine how this substance converts normal cells into cancer mutation. The chief result was cancer of the human skin produced by irritating materials used in varying occupations. In the past five to seven years, the British have been studying carcinogenic activity in nitrogen mustards. These are more readily usable than the hydrocarbons, which are "sluggish and complex." These carcinogens (the mustard group) possess two highly reactive side-chains which affect chromosomes adversely. There is an analogy between the effects of the mustards and that of ionizing radiation. Nitrogen mustard induces anomalies of cell division, and seems to affect the nuclei especially. Cross linking chemical agents were tested biologically and were found to be similar in action to the mustards and to radiation. "We do not know chemically with which of these cell receptors these agents combine, but high in the list of possibilities are the nucleic acids." The giant chromosomes of the salivary glands of the fruit fly, Drosophila, offer favorable biological material for study of cell changes. There is support for an older view that change from the normal to the malignant cell may involve a process of biological loss. It may yet become possible through chemotherapy to determine how the growth of malignant cells can be retarded by treatment with the enzymes in which these cells are defective. There may be a specific biochemical deficiency.

Continuing the discussion, Prof. Geoffrey Hadfield, of the Imperial Cancer Research Fund, Royal College of Surgeons, stated that his theme was carcinogenesis. Malignant disease arises in the protective epithelium, i. e., skin, upper respiratory tract, upper gastrointestinal and lower genitourinary areas. The inorganic salts of arsenic are carcinogenic to man, although experimental animals are not susceptible. The incidence of lung cancer in workers with chromium salts is 16 to 80 times that of the general population. A similar situation exists in those working with crude tar. Cancer of the epithelial tissues is usually slow growing, from 5 to 20 years. The epithelial surface, so constantly subjected to carcinogenic insult, becomes profoundly altered or "initiated," thus undergoing a sort of "promotion." So a weak irritant,

such as croton oil, may produce a trigger effect. The Rhesus monkey shows considerable resistance to these potent carcinogens, coal tar et al. "Cancer arises when such cells are propelled into continuous mitotic division by a promoting agent."

Further discussion was carried on by Dr. P. R. Peacock, of the Cancer Research Department of the Royal Beatson Memorial Hospital in Glasgow. Two main trends in cancer research interest him especially: first, a study of the life of cells as determined by tissue culture, virology, and electron microscopy, coupled with biochemistry and physical chemistry (but only a small group of specialists are working in this field); second, demographic and geographic peculiarities which seem to apply to the incidence of human cancer. In Scandivania cancer of the stomach occurs about twice as often as in Great Britain. Even in the same country, there may be areas of socioeconomic groups where exposed sites, e. g., skin and upper alimentary canal, are susceptible, but not the lungs nor rectum! Industrial and environmental hazards are, of course, well known, if not well understood. Destructive distillation with incomplete oxidation produces 3, 4-benzopyrene. This is found in soot, pitch, and polluted air. It may become lethal. Cooking and smoking give off, probably, carcinogenic vapors. "The association between cigarette smoking and lung cancer seems to be established beyond all reasonable doubt, although the causal action is not yet determined." Under experimentation at this time are mice exposed to soot and the unfiltered air of Glasgow and a control group of mice living in protection from polluted air. Results are to be determined later.

Dr. R. L. Worrall commented on a recent trend in cancer research in attempts to check malignant growth by administering fat antioxidants, such as tocopherols, gallates, and other substances used in preserving foods. The thought is that something is wrong with the fat metabolism in cancer victims. Irradiation which can produce cancer tends to induce a self-perpetuating metabolic disorder. In myeloid leukemia there is an early disappearance of marrow fat, and a malignant growth of white cells in the bone marrow. "One can administer peroxide-forming compounds, such as those found in the volatile oils, in order to set up catastrophic reactions in cancer cells." Such experiments are now under way.

In closing, Professor Haddow said that much work is now going on (in Britain) with metabolic antagonists, with special reference to antifolic, antipurine, and antipteridine agents.

I. W. Voorhees, New York.

Otorhinologic Pathology of Bronchial Asthma. J. M. Ferrando Botet, Ann. oto-laryng. (Paris) 73:546 (July-Aug.) 1956.

The author believes that there is a definite relationship between the pathology in the sinuses, the pharynx, the tonsils, and the deviation of the septum and bronchial asthma. He describes the various types of pathologic conditions found in association with bronchial asthma, the symptomatology of these alterations in each area, and the effects of therapy.

He drew the following conclusions: 1. The otorhinologic morbidity was greater in infants and younger asthmatic adults than in older persons. 2. Tonsillar infections were present in 22% of the asthmatics; this number was definitely decreased following tonsillectomy. However, a number were not influenced by this procedure. 3. Operation on the nasal septum rarely influenced the course of asthma. However, whatever improvement did occur was indirectly due to the improvement of the drainage from the sinuses and thus a decrease in the infections. 4. Thirty-four per cent of the asthmatics showed definite sinus infection, of which number 16% involved the ethmoids, 21% involved the maxillary, and many had multiple sinus infections. He prefers medical treatment of the sinuses rather than surgical intervention.

Persky, Philadelphia.

Books

Local Analgesia: Head and Neck. By Sir Robert Reynolds Macintosh, D.M., F.R.C.S. (Edin.), Nuffield Professor of Anesthetics, University of Oxford, and Mary Ostlere, M.B., Research Assistant, University of Oxford. Illustrated by Marjorie Beck. Price, $6.50. Pp. 138. E. & S. Livingstone, Ltd., 16 and 17 Teviot Place, Edinburgh 1, 1956.

This excellent British book is published in the United States by Williams & Wilkins Company. It follows the Chinese dictum that one picture is worth a thousand words. Hence, the illustrations are excellent. The text is entirely adequate. Successful local anesthesia is dependent upon a thorough knowledge of anatomy, use of the right drug, and management based on previous experience. Technique is, of course, also basic. All of the elements to be considered in local analgesia are well combined in a way which makes the text readable, even enjoyable.

Kurze Geschichte der Vestibularisforschung. By Ernst Wodak, M.D. Price, $3.30. Pp. 162, with 8 illustrations. Georg Thieme Verlag, Herdweg 63, (14a) Stuttgart, 1956.

This small book is written and organized in the time-honored style of A. Politzer's "History of Otology" (1912). It is not actually a text, for instance, like J. J. Fischer's "The Labyrinth," which was also published quite recently (Grune & Stratton, Inc., 1956). Rather, it is a historical review aimed at an audience that knows the vestibular field as such. The author himself has been actively engaged in vestibular research for almost 40 years, first in his native Prag, and later in Tel Aviv. He states in the Introduction that the book was written for the enlightenment of a younger generation and for recollection by the older one. The first pages of the book are devoted to a brief history of this relatively young field of science, starting with Cotungo's anatomical studies (1775) and Purkinje's first contributions to a symptomatology of vertigo (1820) and culminating in the work of R. Bárány, upon which modern vestibular testing was founded. Bárány's first publication on the subject, by the way, last year had its 50th anniversary, being published in 1906. After this review follows, chapter by chapter, an enumeration of vestibular research in Austria, Holland, etc., throughout Europe and the rest of the world. This method of reporting places emphasis not so much upon the contributions of individual researchers but rather upon that of "schools," which at that time were at their summit in Europe, such as the famous Dutch School of Magnus and De Klejn in Utrecht. McNally's Laboratory in Montreal, Canada, might be considered an off-shoot of this latter school on this continent. The entire text is interspersed with anecdotes, mostly of the author's own recollections.

The United States is one of the few countries of which the author did not have a first-hand knowledge, although he had apparently very able help in the preparation of this chapter. Contained in it are some sobering reflections concerning the reasons behind the recent shift in scientific eminence from Central Europe to the United States. The author puts less emphasis upon the drainage of talent from Central Europe due to emigration (which indicates his own personal detachment) than upon the efforts made by those in the United States, which resulted in a surge of scientific development. In his own words: "The former pupils (i. e., Americans in Europe) developed into first-class researchers and became reliable teachers in their own right."

It is a pleasure to recommend this "Brief History of Vestibular Research."

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. G. M. T. Hazen, 208 Canada Building, Saskatoon, Sask.
Secretary: Dr. G. A. Henry, 328 Medical Arts Building, Toronto.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Bronchoesophagological Society

President: Dr. Theodor Hünermann, Düsseldorf.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. Henry M. Scheer, 522 W. End Ave., New York 24.
Secretary: Dr. Louis Savitt, 3402 W. Lawrence Ave., Chicago 25.
Place: Palmer House, Chicago. Time: Sept. 8-12, 1957.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Brazil. Time: 1958.

Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Erling W. Hansen, 90 S. Ninth St., Minneapolis.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Broncho-Esophagological Association

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Fairmont Hotel, San Francisco. Time: May 19-20, 1958.
Place: The Homestead, Hot Springs, Va. Time: March 8-9, 1959.

American Laryngological Association

President: Dr. LeRoy A. Schall, 243 Charles St., Boston 14.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Washington, D. C. Time: May 3, 1957.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Percy E. Ireland, 100 College St., Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.

SECTIONS:

Eastern.—Chairman: Dr. Joseph Sullivan, Toronto, Canada.
Southern.—Chairman: Dr. Mercer G. Lynch, New Orleans.
Middle.—Chairman: Dr. Paul M. Moore, Cleveland.
Western.—Chairman: Dr. Sigurd v. Christierson, San Francisco.

American Otological Society, Inc.

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37.
Secretary-Treasurer: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Place: Fairmont Hotel, San Francisco. Time: May 17-18, 1958.
Place: The Homestead, Hot Springs, Va. Time: March 13-14, 1959.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Joseph Gilbert.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

American Society of Facial Plastic Surgery

President: Dr. Irvin J. Fine, 505 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago. Time: October, 1957.

Otosclerosis Study Group

President: Dr. Joseph A. Sullivan, 174 St. George St., Toronto 5, Canada.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.
Place: Palmer House, Chicago. Time: Oct. 12, 1957.

Society of Military Otolaryngologists

President: Col. Wendell A. Weller.
Secretary-Treasurer: Major Stanley H. Bear, M. C., 3810th USAF Hospital, Maxwell AFB, Ala.

Scientific Exhibits

Mobilization of Stapes for Otosclerotic Deafness

SAMUEL ROSEN, M.D., New York

The Scientific Exhibit depicting the mobilization of stapes for otosclerotic deafness was awarded the Hektoen Gold Medal for Original Investigation at the 1956 summer meeting of the American Medical Association.

The exhibit was designed to present clinical and historical data, as shown in the following pages. Specially treated cadaver specimens were mounted on the main panel (above) to enable physicians to study the main steps of the technique through the otoscope. Instruments designed by me were shown in the order of their use in the procedure.

For the patient mobilization is a minor operation, but for the surgeon it demands a high degree of skill and should be performed only by those trained in endaural surgery. Practice on at least 50 fresh cadavers is recommended because of the great number of anatomical variations encountered. It is also essential that careful audiologic work-up be made preoperatively to determine the suitability of the patient.

The photographs, tables, and audiograms present the salient facts about the Rosen technique of mobilization of the stapes.

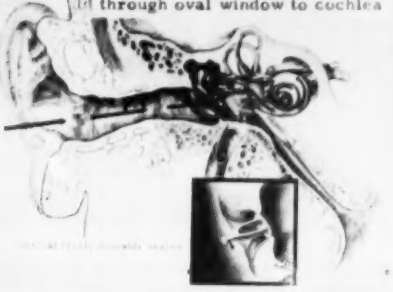
Accepted for publication Sept. 13, 1956.

Consulting Otologist, and Chief of Mobilization Clinic, The Mount Sinai Hospital.

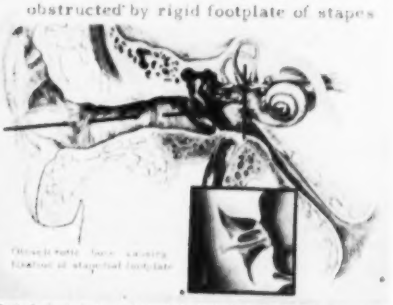
Shown as a scientific exhibit of the Section on Laryngology, Otology and Rhinology at the 105th Annual Meeting of the American Medical Association, Chicago, June 11-15, 1956.

MECHANISM OF DEAFNESS IN OTOSCLEROSIS

Normal sound transmission through oval window to cochlea



Sound transmission to cochlea obstructed by rigid footplate of stapes



INDICATIONS FOR STAPES MOBILIZATION

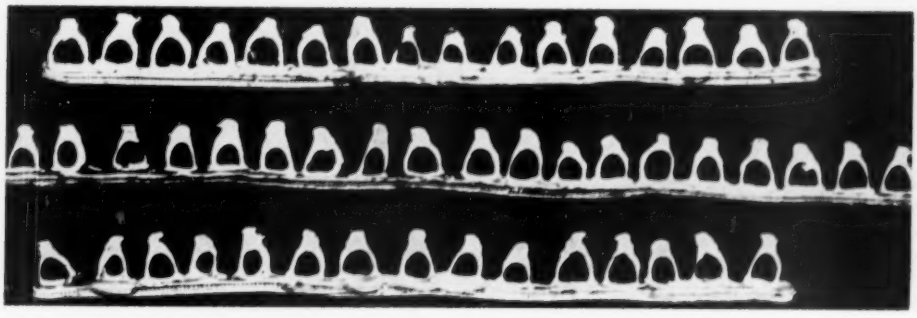
1. All cases suitable for fenestration.
2. Cases of early otosclerosis not yet operable by fenestration technique.
3. Cases of profound otosclerotic deafness where mobilization can contribute to auditory rehabilitation with or without the hearing aid.

ADVANTAGES OF STAPES MOBILIZATION

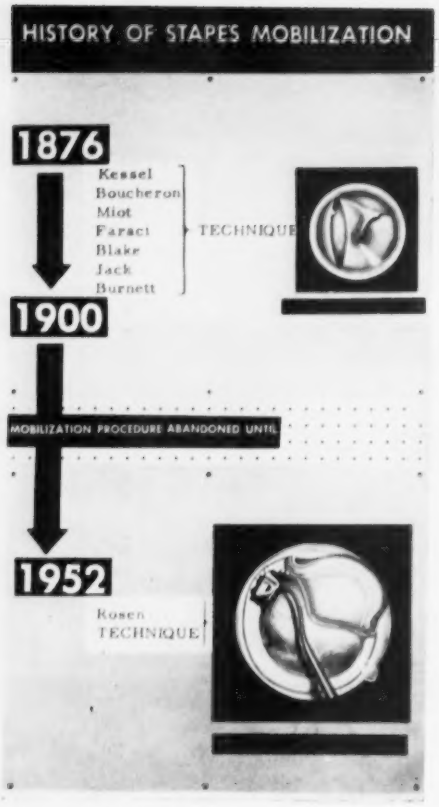
1. Operation performed without pain under local anesthesia through ordinary ear speculum.
2. No shock.
3. Hospitalization - 24 hours. Resumes work in 1 or 2 days.
4. Post-operative treatment - none.
5. Patient may swim, dive, travel by air, etc.
6. Mobilization failure may be followed by successful fenestration.

COMPLICATIONS

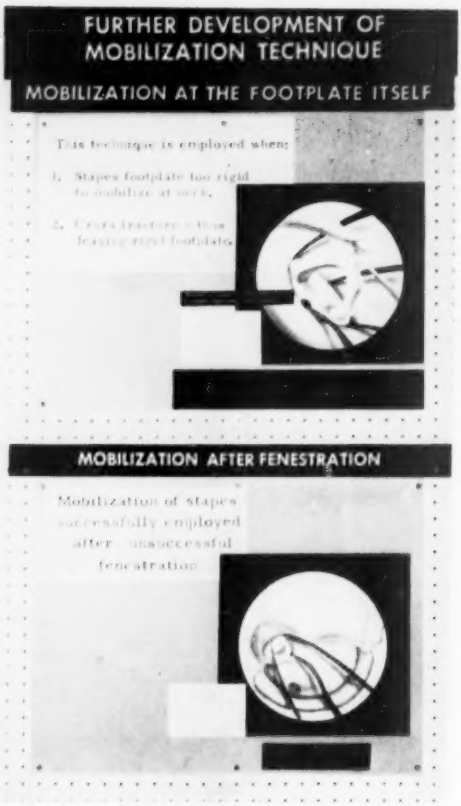
1. Otitis media - rare.
2. Persistent perforation of drum - rare.
3. Facial paralysis - none.
4. Labyrinthitis - none.



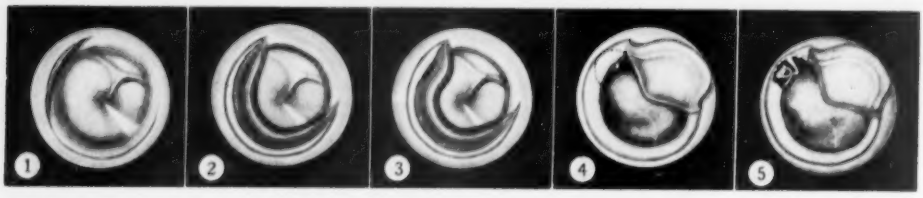
Cadaver specimens showing variation in structure of stapes.



Anatomical drawing at top shows myringotomy opening used in earlier attempts at mobilization. This opening did not permit sufficient visualization of the stapes. Bottom drawing shows Rosen technique, which permits complete visualization of structures. Mobilizer shown at neck of stapes.

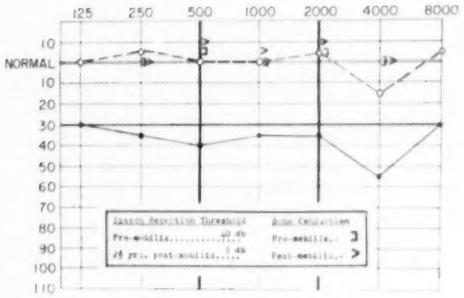

FURTHER DEVELOPMENT OF MOBILIZATION TECHNIQUE
MOBILIZATION AT THE FOOTPLATE ITSELF
MOBILIZATION AFTER FENESTRATION
Mobilization of stapes successfully employed after unsuccessful fenestration

Anatomical drawing at top shows footplate mobilized, with sharp-pointed explorer inserted between bony wall of oval window and footplate. The fenestra made through the oval window at the peripheral margin of the footplate allows sound to enter the internal ear directly. Hearing by this method has been improved. Bottom drawing shows incus removed at fenestration operation and the rigid stapes being mobilized at the footplate.

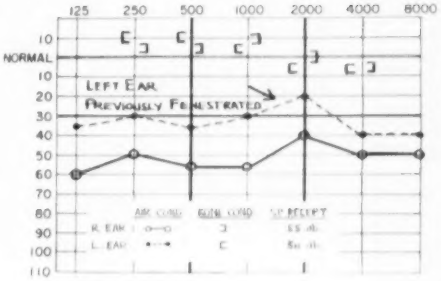


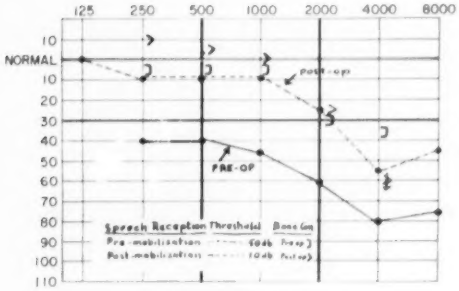
Steps of Technique: 1. Incision of skin from 12 to 3 o'clock. 2. Elevation of skin to drum margin. 3. Initial opening into tympanum. 4. Drum elevated, thus exposing tympanic cavity with inadequate visualization of incus and stapes. 5. Bony posterior canal wall removed for complete visualization of ossicles.

ACHIEVEMENTS OF MOBILIZATION

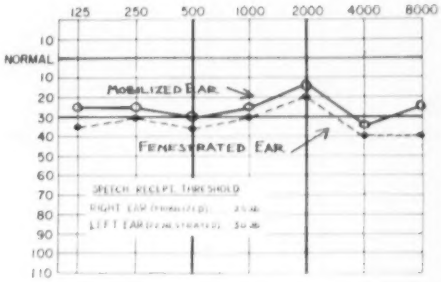


Restoration of Normal Hearing: Normal hearing four years after mobilization.

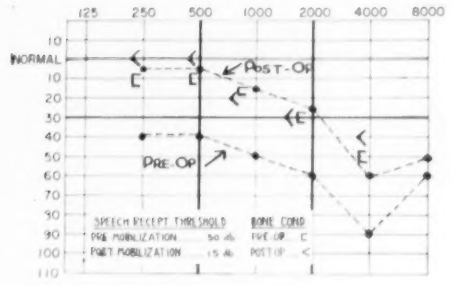




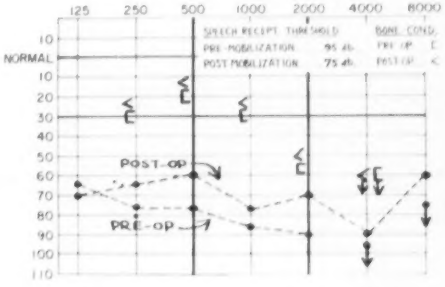
Almost Normal Hearing: Near normal hearing for speech despite some cochlear degeneration two and one-half years after mobilization.



Restoration of Useful Binaural Hearing: Above, premobilization. Below, binaural hearing after mobilization.



Restoring Conductive Component (Stapedial) in Mixed Deafness (Cochlear and Stapedial): Closing air-bone gap. Full utilization of cochlear potential.



Permitting Successful Use of Hearing Aid in Extreme Deafness: Since mobilization is able to hear average conversation with hearing aid. Before mobilization could not.

Results of Mobilization

1. Total Reaching 30 Db. or Better	32%
A Cases (Preop maximum bone-conduction loss 10 db.)	48%
B Cases (Preop. maximum bone-conduction loss 20 db.)	34%
C Cases (Preop. maximum bone-conduction loss 30 db.)	21%
2. Hearing Improved to Various Levels of Usefulness	
10 db. or more improvement	57%
15 db. or more improvement	42%
20 db. or more improvement	30%
3. Improved by Mobilization at the Footplate (When mobilization could not be achieved at the stapedial neck, mobilization at the footplate at once achieved the following additional results)	
Improved to 30 db. or better	34%
Improved to various levels of usefulness	55%
4. Revisions	
Improved to 30 db. or better	25%
Improved to various levels of usefulness	40%
5. Improved Following Mobilization but Later Regressed to Preoperative Level	1.7%
6. Hearing Worse than Preoperative Level Following Mobilization	1.4%

Summary

Results show that in suitable cases of deafness due to otosclerosis normal hearing can be restored by mobilization of the stapes. In addition, the mobilization operation can be successfully performed in a much wider spectrum of deafness due to otosclerosis than was hitherto possible. Mobilization of the stapes should be the first procedure of choice in all cases which can be benefited surgically.

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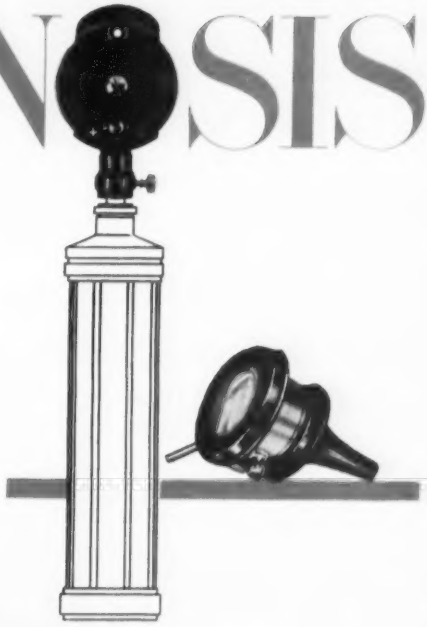

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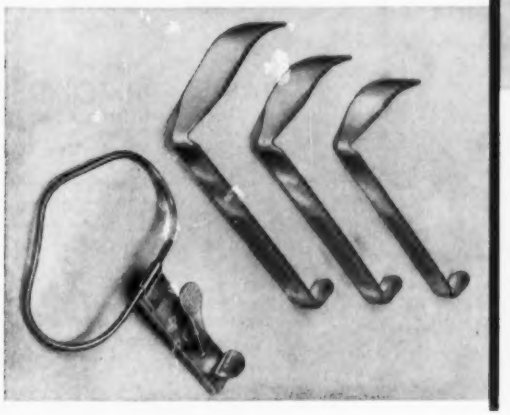

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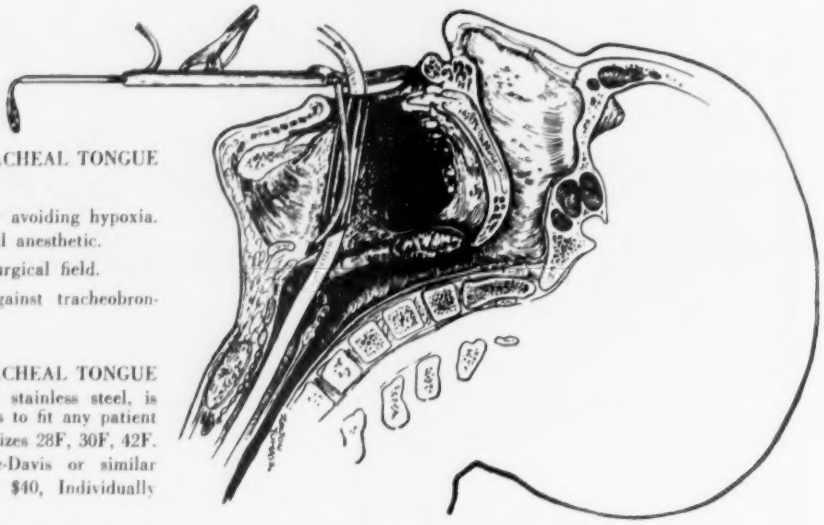

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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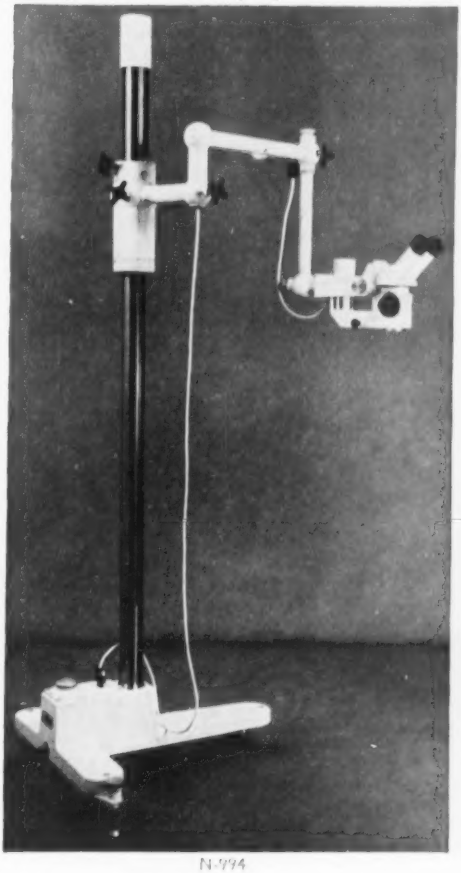

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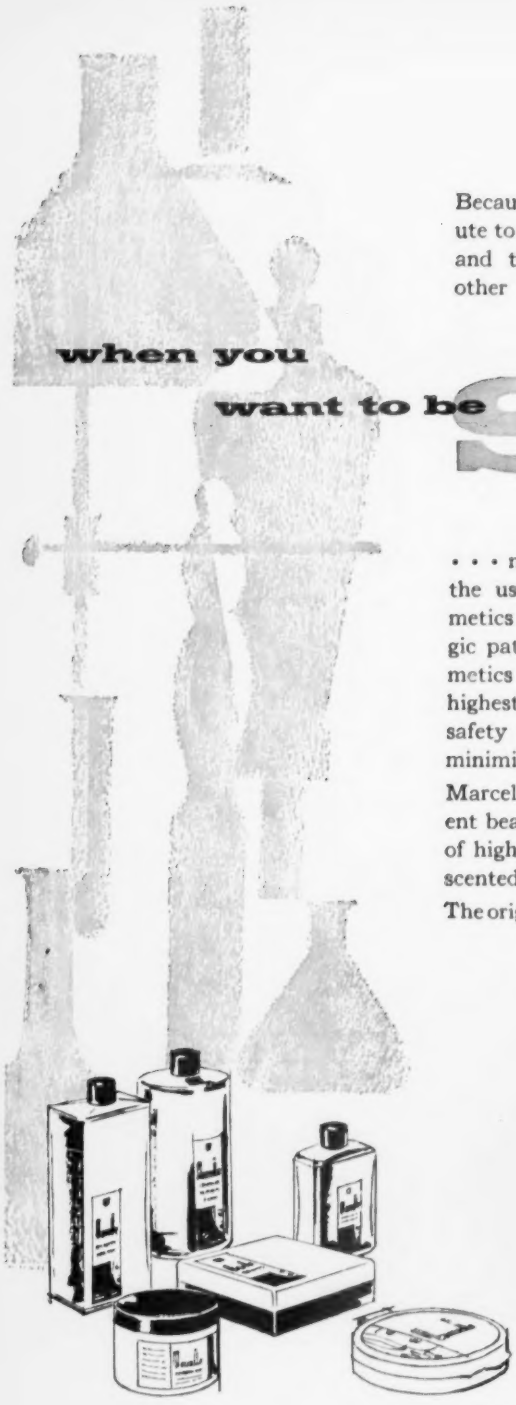

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